SOME OF THE COMPLICATIONS OF SCARLET FEVER, OCCURRING AFTER THE PRIMARY INFECTION.

THESIS FOR THE DEGREE OF M.D.

by

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I have been Assistant Medical Officer in the Sheffield City Hospital for Infectious Diseases from 1st August 1904 to 31st December 1906. During this period Scarlet Fever had been epidemic in Sheffield, the admissions numbering:-

1904	August 1st to December 31st	1180
1905		2219
1906		3104
		6503

Over one third of the cases have been under my care, and I have had the opportunity of seeing any case in the hospital that exhibited any feature of unusual interest. The first subject I propose discussing, is some of the rare complications of Scarlet Fever occurring particularly, at that most critical period of the disease, between the 18th and 28th day, counting from the day of onset.

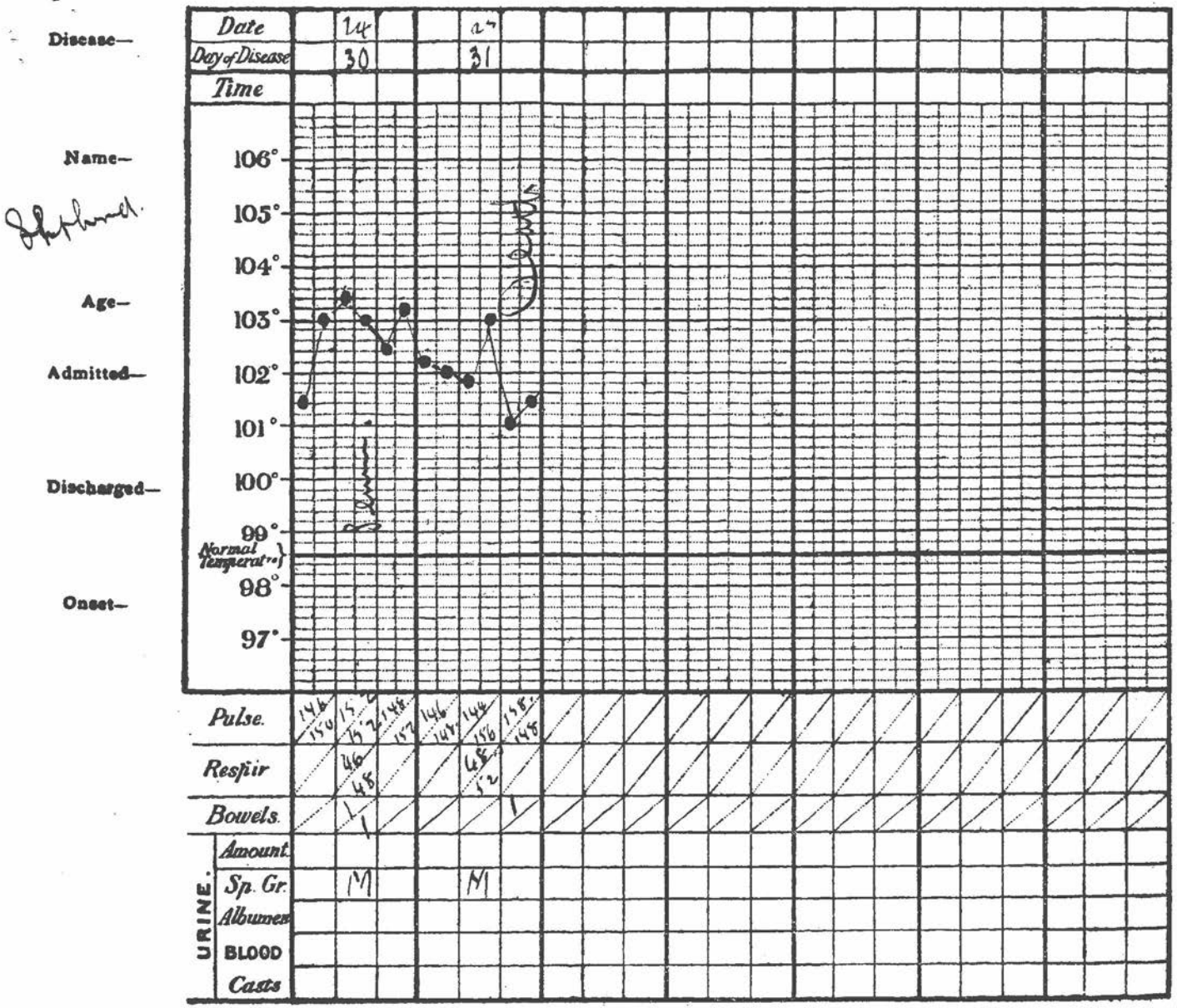
The ordinary simple attack of scarletina cannot be said in its initial stage, to be an infection of great severity. After the early symptoms, the temperature returns to normal in a day or two, the throat symptoms pass away, and in a large majority of cases the convalescence is uneventful. But during this time morbid processes are going on, that may perhaps only manifest themselves in a slight degree/

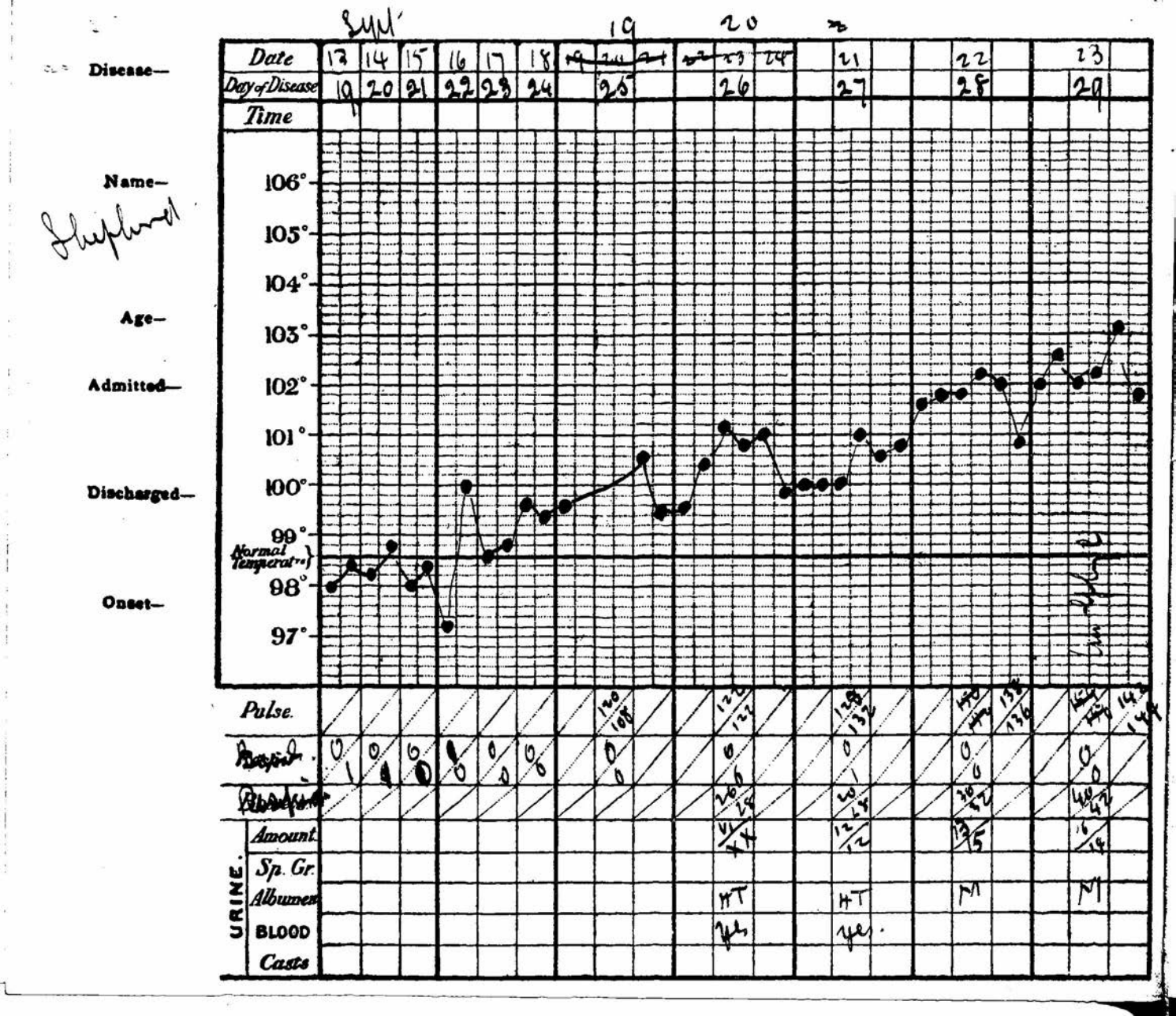
degree of albuminuria, or recrudescence of the early cervical adenitis; on the other hand,we may get complications of the most alarming description.

There is a tendency amongst recent writers to look on many of the complications appearing at this period as a secondary infection, probably of a streptococcal character. It is my intention to give the details of some cases that appear to bear out this idea, and then to discuss what literature I have been able to find throwing any light on the subject.

The complication known as "secondary throat" or "secondary tonsillitis" is well recognised in most of the larger text-books on infectious diseases, but it is scarcely mentioned in the books on general medicine.

It occurred in 14 out of the 2219 cases treated at Lodge Moor Hospital in 1905. The condition is usually not dangerous,although the symptoms may cause some alarm both to the patient and the medical attendant, there is marked swelling and injection of the fauces generally, often some soft exudation on the tonsils, the cervical glands are enlarged, the temperature is moderately elevated; as a rule in a day or two the symptoms abate and the recovery is rapid. In one or two of the cases I have seen, there/


Disease—
Name—
Age—
Admitted—
Discharged—
Onset—
Date
Day of Disease
Time
14
30
27
31
106°
105°
104°
103°
102°
101°
100°
99°
Normal Temperature
98°
97°
Pulse.
Respir
Bowels.
URINE.
Amount
Sp. Gr.
Albumen
BLOOD
Casts


Disease—
Name—
Age—
Admitted—
Discharged—
Onset—
Date
Day of Disease
Time
106°
105°
104°
103°
102°
101°
100°
99°
Normal Temperature
98°
97°
Pulse.
Respir.
Bowels
URINE.
Amount
Sp. Gr.
Albumen
BLOOD
Casts

Aug. Sept.

Disease—

Name— Rebecca Stephens

Age—

Admitted—

Discharged—

Onset—

Date	30	31			1			2			3		4	5	6	7	8	9	10	11	12
Day of Disease								8			9		10	11	12	13	14	15	16	17	18
Time	9	6	10	2	6	10															

106° 105° 104° 103° 102° 101° 100° 99° (Normal Temperature) 98° 97°

Typhoid Pneumonia

Pulse.																					
Respir																					
Bowels.		0			1 0			1 1			0 1		0 1	0 0	0 1	0 1	0 1	0 1	0 1	0 1	0 1
URINE. Amount.																					
Sp. Gr.																					
Albumen					Trace			Trace						nil		nil		nil			Trace
BLOOD																					
Casts																					

there was moderate delirium; the swelling of the faucial structures may cause some difficulty in respiration. The cases I wish particularly to refer to occurred during the two and a half years I have been here, in them the throat symptoms were particularly severe, and they were accompanied by other evidences of a general septic infection.

The notes of the casee are as follows:-

I. R.S. Female aged 20 years.

First fell ill on August 26th 1904 with the usual symptoms of headache, sore throat, vomiting and general punctiform rash on the 27th. She was admitted to hospital on August 31st.

August 31st. Fauces:- injected, tonsils swollen with slight gollicular exudation. Tongue:- well marked strawberry. Skin:- Bright, general punctate rash. There is some cervical adenitis on both sides. Temperature 99.2°F. Patient is fairly comfortable.

The case was a quite definite one of mild scarlet fever, the temperature after admission never reaching higher than 99.6°. The patient was allowed up on the 16th day of disease, and no further notes were taken until September 16th the 22nd day of disease.

September 16th. Temperature 100°F, cervical adenitis left side slight, several small papular swellings on the arms/

the arms and legs, especially marked on the extensor aspects of the elbows and knees, one or two on the face. The papules are about the size of a small pea, they are quite hard, and are painful to the touch.
September 17th. All the papules have central haemorrhages and are rather more numerous than yesterday, otherwise the condition is much the same.
September 18th. Patient complains of pains in the limbs, the pain does not appear to be localised to any particular joint. Papules are increasing in number and size, they do not appear to contain any pus. The gums are red, swollen and tender, there is commencing ulceration behind the upper and lower incisors close to the teeth. An exudation, of a thick membranous or pultaceous appearance, is present especially on the right tonsil. The breath is offensive. Cultivation of the throat shews cocci, streptococci being very numerous.
September 19th. Several more haemorrhagic spots have appeared, old ones are drying up. The ulceration of the gums is spreading round the alveolar margin close to the teeth. Exudation on the tonsils is more marked. Auscultation reveals occasional cardiac irregularity but no murmurs. Pain is referred definitely to the elbow and knee joints, there is no swelling./

swelling. Slept well through the night and taking nourishment freely.

September 20th. Ulceration of gums increasing. Complains of left leg being asleep, otherwise no change.

September 21st. The gums are falling away from the teeth especially behind the upper and lower incisors, the teeth are loosening. Profuse offensive discharge from the mouth and nose. The membranous deposit is separating from the tonsils and leaving deep ulcers. Complains of loss of sensation in left little finger and half of ring finger.

September 22nd. Taking nourishment well, but very restless at night. Ulceration of the mouth and throat still continues to spread. Breath very offensive. Heart regular, no murmurs.

September 23rd. Ulceration still spreading, it is now very marked on the pharyngeal wall.

The condition of the patient became rapidly worse, many of the loosened teeth came out, the lower jaw in particular became denuded of gum. She died on the morning of September 26th.

During the last few days, blood and albumen were present in the urine, but as menstruation was going on the significance of the fact was overlooked.

Autopsy/

Autopsy. September 27th.

Body well nourished. Rigor mortis marked. Numerous spots, some of which have undergone ulceration and varying in size from a small pea to a sixpence, are scattered somewhat symmetrically over the body; there are three on the face, about a dozen on each leg and foot in the region of the ankle, a few round each knee, several on the back especially in the sacral region, and about three or four on each hand and wrist.

Respiratory system. Ulceration and thickening of the mucous membrane of the upper part of the larynx, involving especially the free edges of the epiglottis and the epiglottidean folds. Some thickening of the vocal cords, but no definite reduction in lumen. Trachea injected, the mucous membrane covered with thick mucous secretion, there is nothing suggesting membrane. The pleural cavities are normal. There are numerous and extensive patches of broncho-pneumonia at the bases, and along the posterior aspects of both lungs.

Digestive System. Very extensive ulceration of the fauces and pharynx, spreading downwards round the opening of the larynx and upper part of the oesophagus, some of the ulcerated surfaces are covered with dirty white slough.

Stomach/

Disease—

Name— Arnold Lupe.

Age—

Admitted—

Discharged—

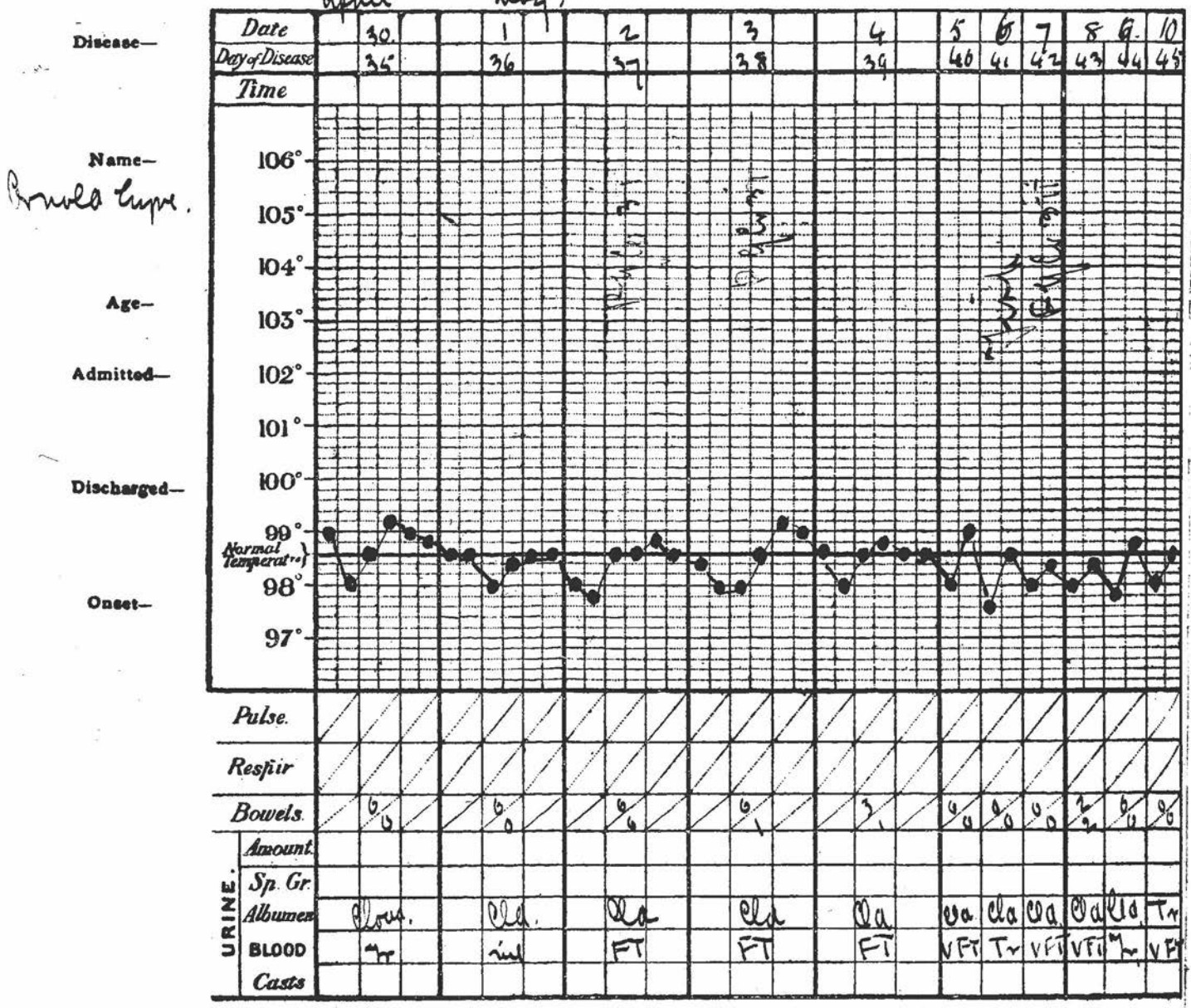
Onset—

May.

Date	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25					
Day of Disease	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60					
Time																				

106° 105° 104° 103° 102° 101° 100° 99° Normal Temperat^re 98° 97°

Pulse.																				
Respir																				
Bowels.	0 0	0 0	1 1	1 0																
URINE. Amount.	14½	19/20	14½	16/20	16	16/20	16													
Sp. Gr.																				
Albumen	ppt.	ppt	ppt	ppt	ppt	ppt	ppt	ppt								Discharged				
BLOOD	ppt.	nil	ppt	V PT	nil	V PT	V PT	V PT	nil	nil										
Casts																				


April
May.
Disease—
Date
Day of Disease
Time
30.
1
2
3
4
5
6
7
8
9
10
35
36
37
38
39
40
41
42
43
44
45
Name—
Bowel type.
Age—
Admitted—
Discharged—
Onset—
106°
105°
104°
103°
102°
101°
100°
99°
Normal Temperature
98°
97°
Pulse.
Respir
Bowels.
URINE.
Amount.
Sp. Gr.
Albumen
BLOOD
Casts

Disease—

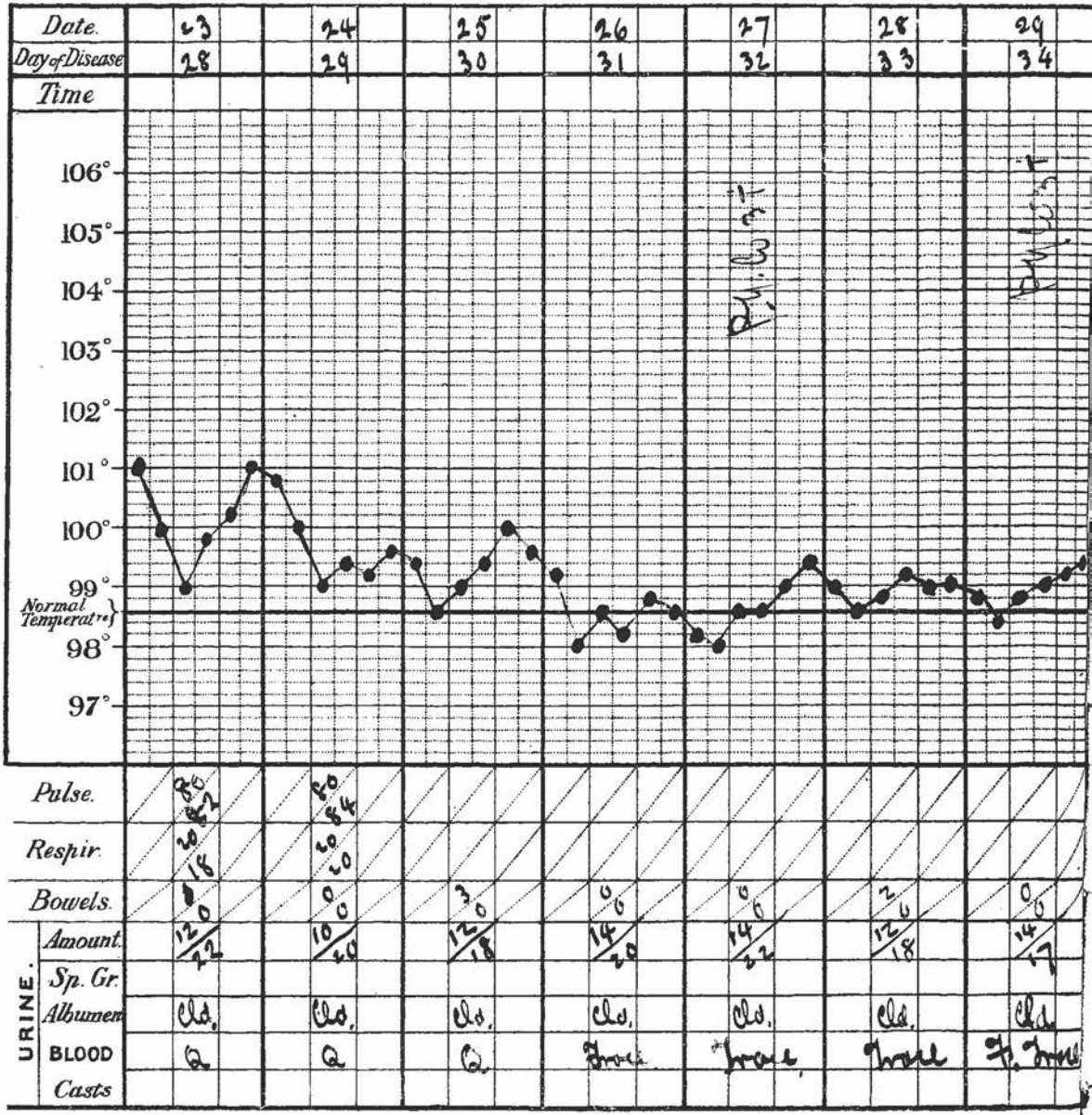
Name—
Arnold Lyne.

Age—

Admitted—

Discharged—

Onset—



Disease—

Name—

Age—

Admitted—

Discharged—

Onset—

April

Date		16			17			18			19			20			21			22		
Day of Disease		21			22			23			24			25			26			27		
Time	2 6	10 2	6 10	2 6	10 2	6 10	2 6	10 2	6 10	2 6	10 2	6 10	2 6	10 2	6 10	2 6	10 2	6 10	2 6	10 2	6 10	
Pulse.					96			98			104			104			94			100		
					100			100			100			80			92			104		
Respir					22			20			20			20			24			22		
					22			22			20			18			22			22		
Bowels.		0			0			0			0			0			2			0		
		0			0			1			0			0			0			0		
URINE. Amount																						
Sp. Gr.																						
Albumen		Trace			Trace.			Clouds.			Clouds.			Clouds.			Clouds			Clouds		
BLOOD								Granular			0			0			0			0		
Casts																						

Disease—

Name—

Age—

Admitted—

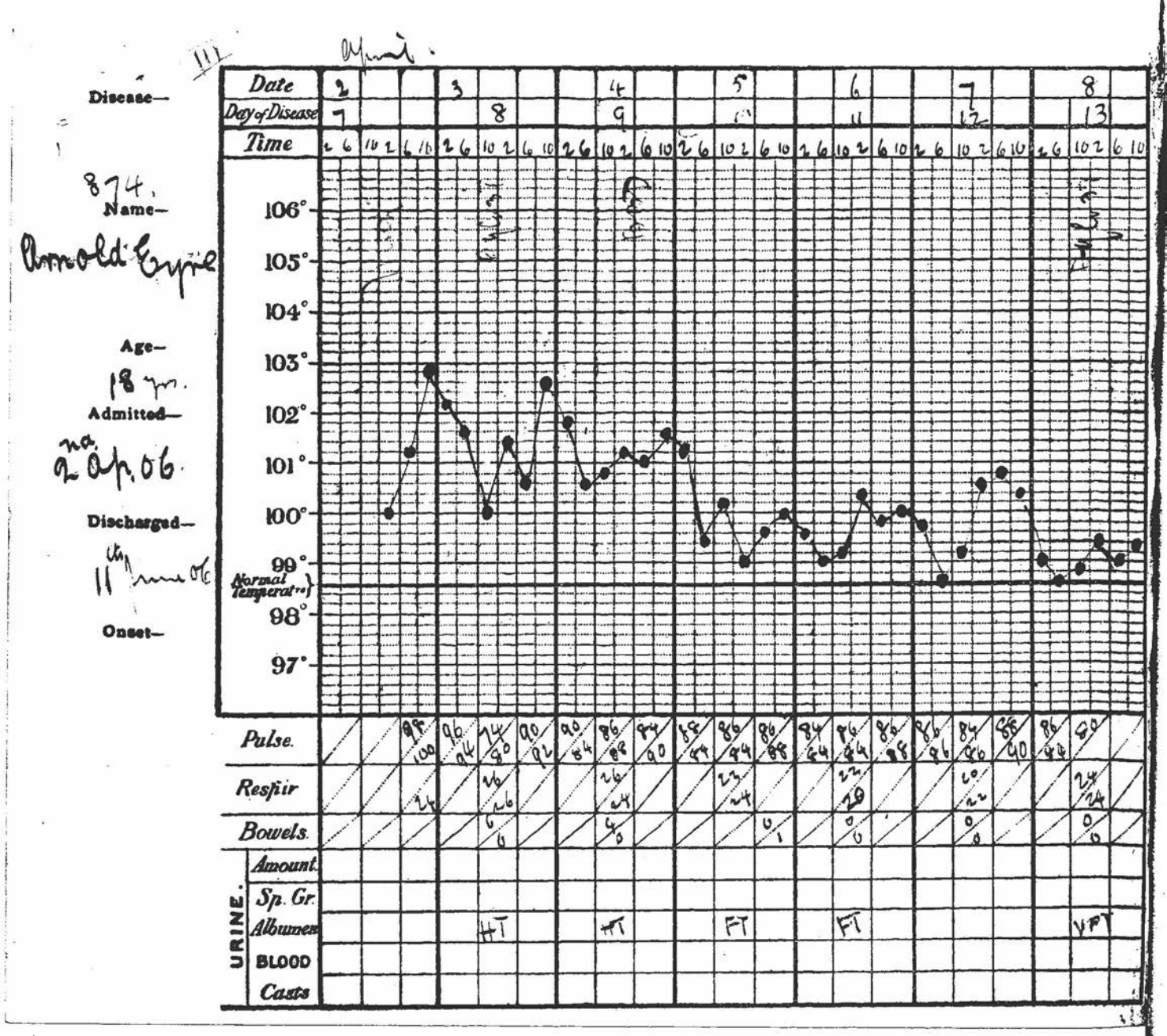
Discharged—

Onset—

Date	9	10	11	12	13	14	15
Day of Disease	14	15	16	17	18	19	20
Time							

106° 105° 104° 103° 102° 101° 100° 99° Normal Temperature 98° 97°

Pulse.							
Respir							
Bowels.	0/2	0/1	0/2	1/0	6/0	0/1	1/0
URINE. Amount.							
Sp. Gr.							
Albumen	NFT	FT	Trace.	Trace.	Trace.	FT	Trace.
BLOOD							
Casts							


Disease—
874.
Name—
Arnold Eyrie
Age—
18 yrs.
Admitted—
2 Oct. 06
Discharged—
11th June 06
Onset—
Date
Day of Disease
Time
106°
105°
104°
103°
102°
101°
100°
99°
Normal Temperature
98°
97°
Pulse.
Respir
Bowels.
URINE.
Amount.
Sp. Gr.
Albumen
BLOOD
Casts

Stomach, ducdenum and jejunum are normal, In the lower 4 feet of the ileum, there are a number (about 20) of small circular punched out ulcers, ranging in size from a pea to a sixpence; they extend down to the muscular coat, the edges are not thickened nor undermined, an area of marked injection surrounds each, the floor of the ulcers is clean, the sites appear to correspond with that of solitary follicles. The peritoneum is normal. Nothing noteworthy about the remainder of the alimentary canal.

Circulatory System. Pericardium, Cardiac muscle and valves appear healthy.

Spleen somewhat enlarged, dark, pulpy, and friable.

Kidneys. The pyramids are perhaps somewhat congested, otherwise nc other naked eye change.

II. A. E. Male. aged 18 years.

First fell ill on March 26th 1906, with the usual initial symptoms. He was admitted to Hospital on April 2nd, when he was found to be suffering from an attack of scarlet fever of moderate severity. The rash was quite well marked.

April 3rd. Heart and lungs normal. Complains of sharp pain in the 6th right intercostal space outside the/

the nipple line, on taking a deep breath. There is some soft exudation on the tonsils.

April 4th. Complains of pain in both shoulders, examination reveals nothing, the pain in the chest has gone.

April 8th. Fauces still somewhat injected, there is no deposit. The pains are quite better. Examination of the heart reveals nothing.

The temperature which had been 102.8° on admission, gradually fell and reached the normal line on April 10th. No more notes were taken until April 14th, the 19th day of disease.

April 14th. Temperature 99.6°. Complains of sore throat, there is some injection and swelling of the tonsils, and some painful adenitis on the right side of the neck.

April 15th and 16th. There is not much change in the patient's condition. The fauces are still injected, and the exudation is perhaps more marked.

April 17th. Definite membranous deposit on both tonsils and on the uvula. The deposit is thick, white and might be described as being of a pultaceous character. It leaves a bleeding surface on removal. The membrane is firmly adherent and seems unusually thick for diphtheritic membrane.

Antitoxin 6,000 units, injected.

Cultivation/

Cultivation on blood serum gives almost pure culture of streptocccci.

April 18th. Temperature rising. Quantity of blood and albumen in the urine. The exudation on the tonsils is still more marked. The breath is very offensive, but the odour does not suggest diphtheria, it is more like gangrene. There is commencing ulceration of the tongue. Some difficulty in swallowing. One or two of the back molar teeth are decayed, the teeth generally are pretty good.

April 19th. Condition worse, patient very ill indeed. Papules with haemorrhagic summits have appeared over the buttocks, the elbows, and the knees. There are subcutaneous haemorrhages, about the size of a shilling, over both external malleoli, and in front of the right knee. Patient slept badly, owing to mucous collecting in the throat.

April 20th. Condition of patient unchanged.

April 21st. Papules drying up. There is now some ulceration of the soft palate, covered with thick white membranous deposit. Blood and albumen are still present in the urine. Otherwise condition unchanged. Temperature running between 101° and 102°.

April 23rd. Deposit clearing away and leaving distinct ulcers. Tongue healing. Eruption over the joints improving, dry scabs forming. Urine still contains quantity/

October

Disease—

Name—

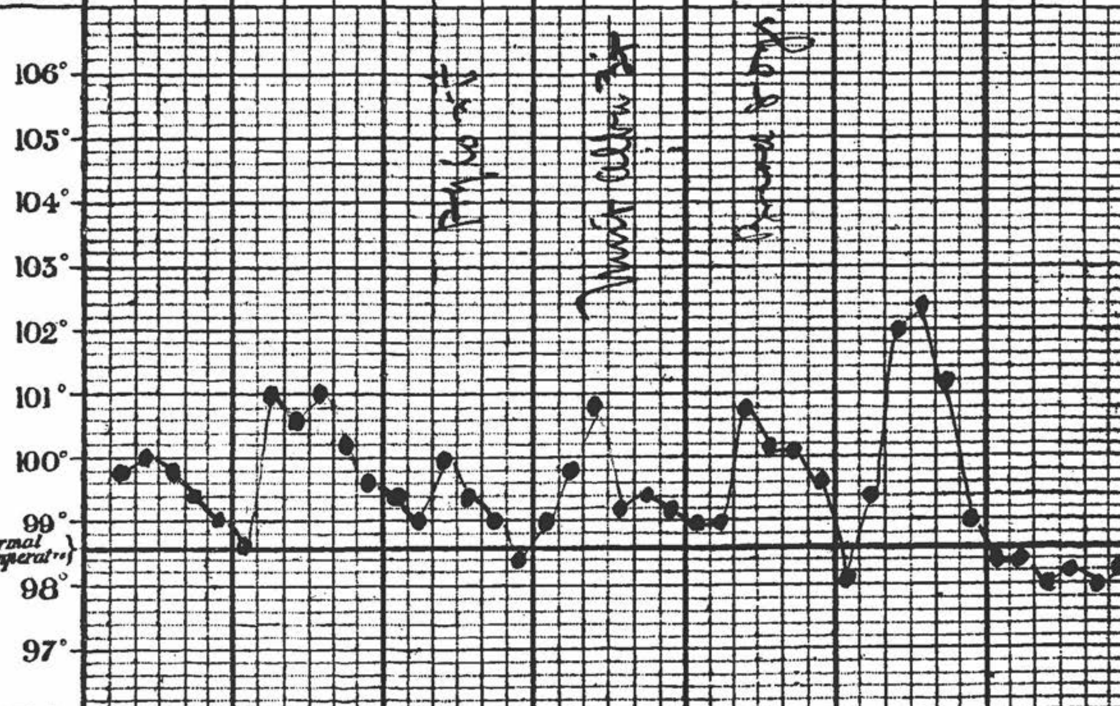
Age—

Admitted—

Discharged—

Onset—

Date	24	25	26	27	28	29	30
Day of Disease	44	45	46	47	48	49	50
Time							



	24	25	26	27	28	29	30
Pulse.	120 / 116	112 / 110	116 / 110	114 / 112	120 / 118	102 / 116	96 / 100
Respir	20 / 20	16 / 16	14 / 14	16 / 14	20 / 16	16 / 14	16 / 16
Bowels.	1 / 0	0 / 0	1 / 1	1 / 1	0 / 1	0 / 1	0 / 1
URINE. Amount.	45 oz.	46 oz.	30 oz.	34 oz.	40 oz	40 oz	39 oz.
URINE. Sp. Gr.							
URINE. Albumen	Cloud	Cloud	Cloud.	Hy trace		H. trace	Trace.
URINE. BLOOD	Trace	Trace	Trace.	Fy trace.		Nil	Nil
URINE. Casts	·2	·1	·2	·1			

[illegible] albumen Esbach.

October.

Disease—

Name—
Arthur Arnold.

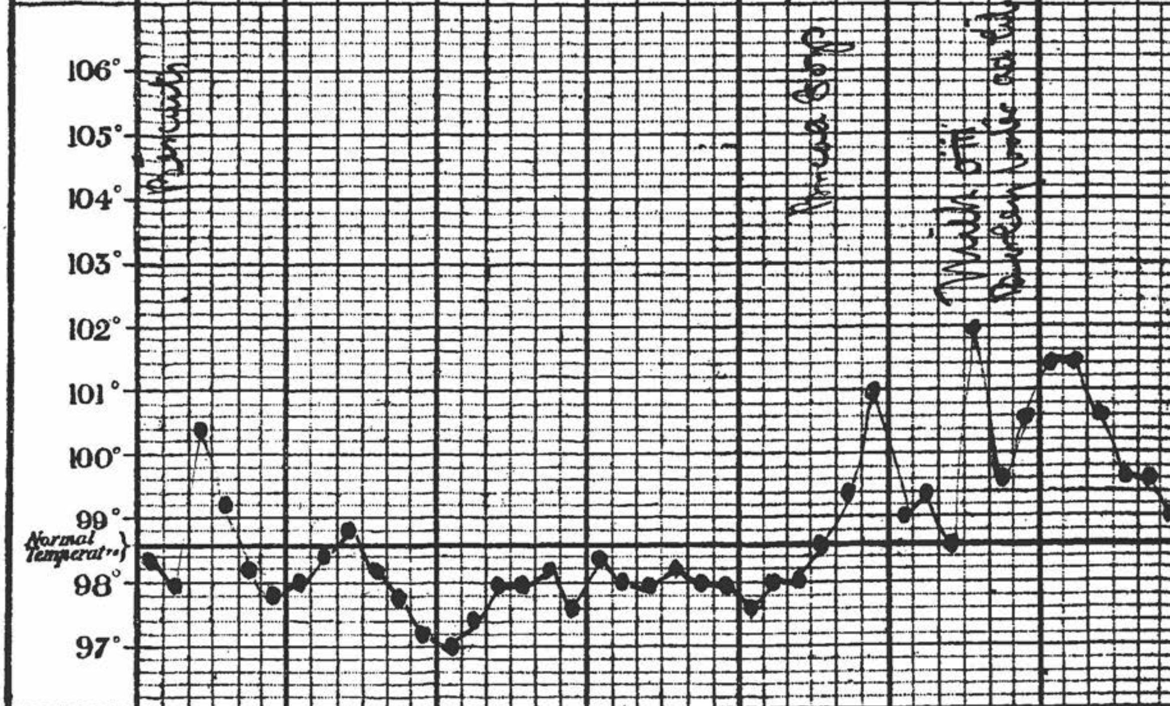
Age—

Admitted—

Discharged—

Onset—

Date		13			14			15		1	16	17	18	19	20	21	22			28	
Day of Disease		33			34			35			36	37	38	39	40	41	42			43	
Time																					



Pulse.		104/106			102/98			92/94			94/92						120/110			114/130	
Respir		14/14			14/12			14/16			14/14						20/20			26/18	
Bowels.		1/0			0/1			0/0			0/1		0/0	0/1	0/1	0/1	1/0	0/0		0/0	
URINE. Amount.																					
Sp. Gr.																					
Albumen		H. trace.			H. trace.			Cloud.			H. Trace.		T	T	T	T	HT	Cloud		Cloud.	
BLOOD		nil			nil			nil			nil		nil	nil	nil	nil	trace.	trace		trace	
Casts		·2						·1									·1			·2	

October 1906.

Disease—

Name— Arthur Arnold

Age— 18 yrs

Admitted—

Discharged—

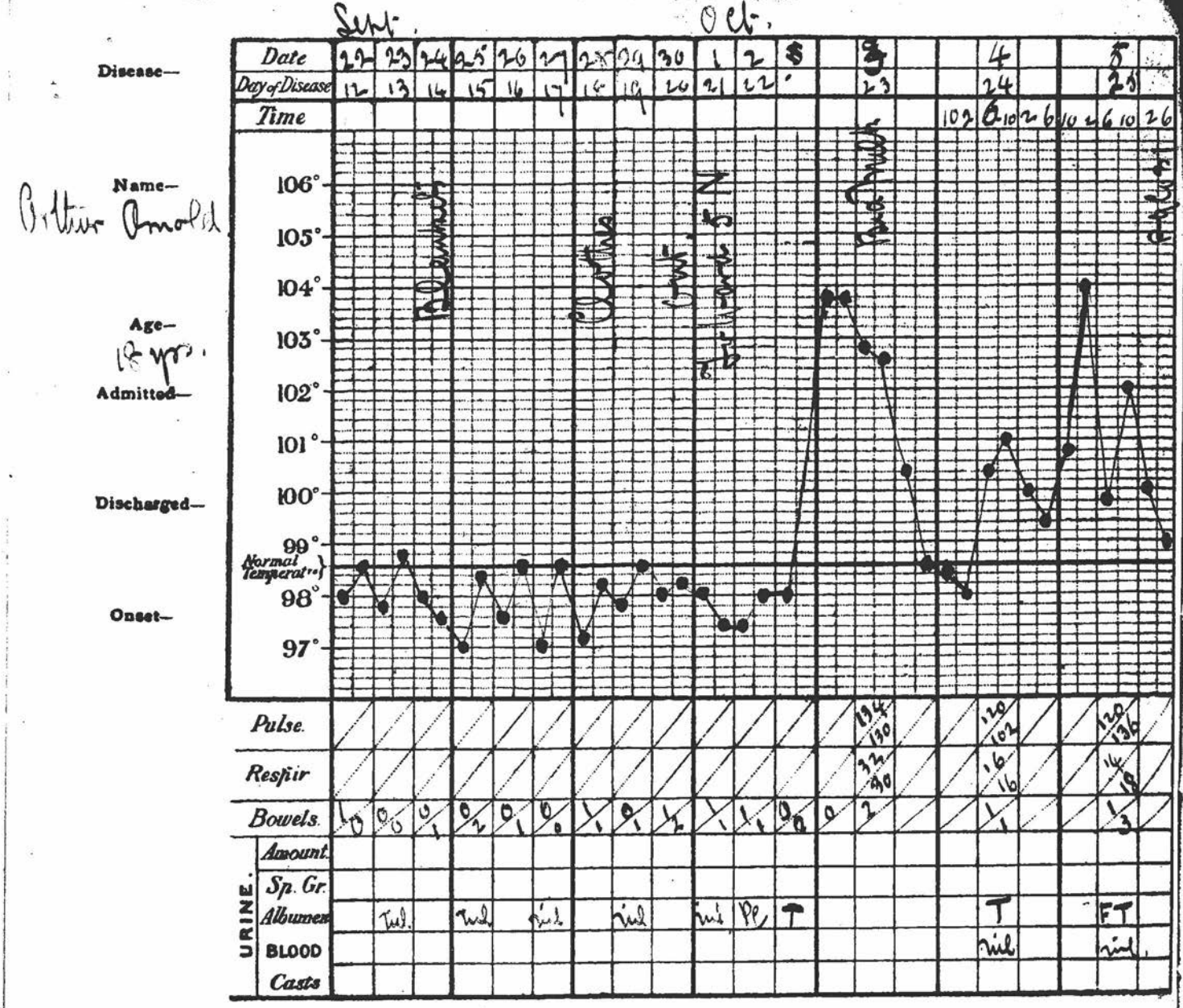
Onset—

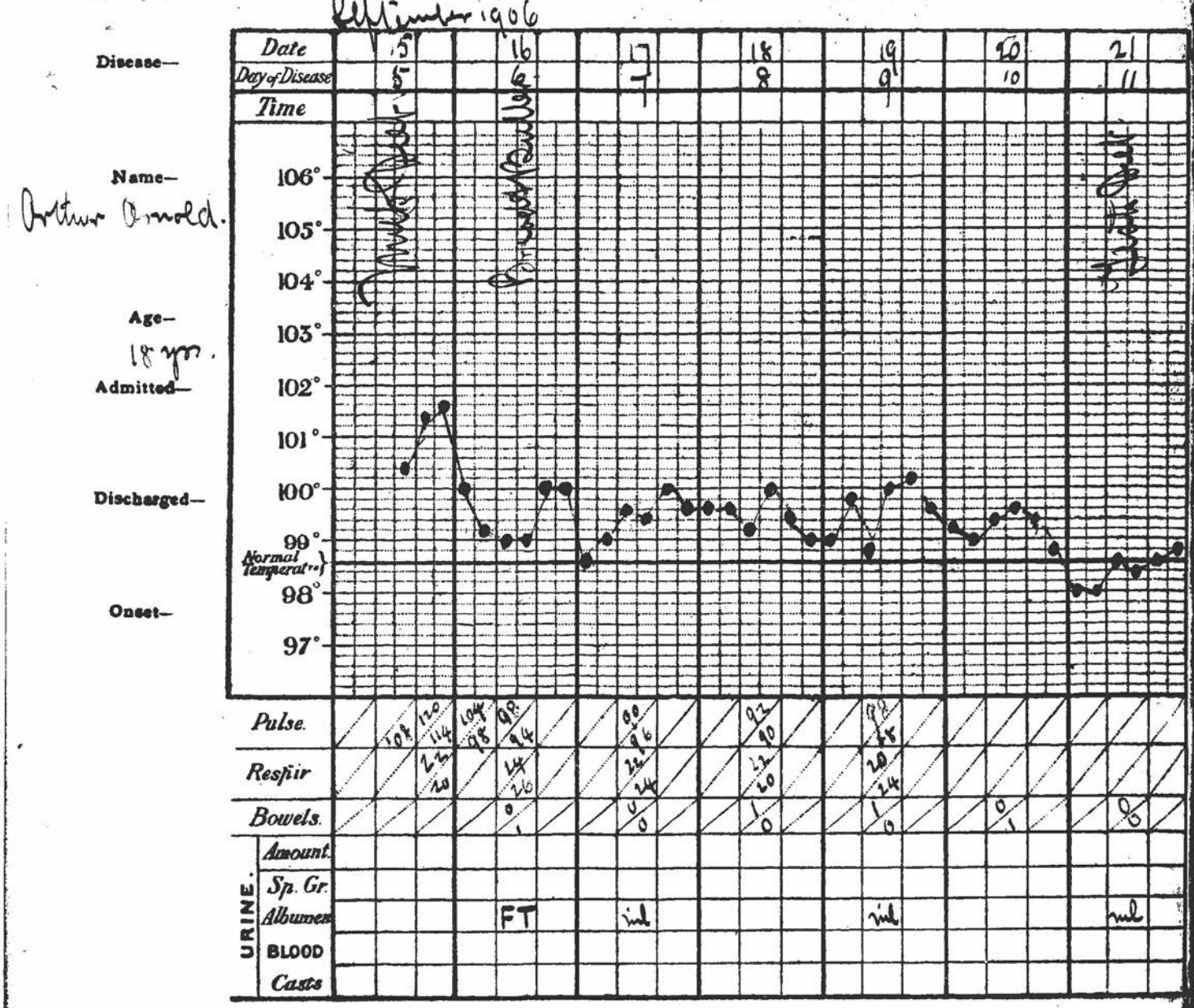
Date	6	7	8	9	10	11	12
Day of Disease	26	27	28	29	30	31	32
Time	10 2 6 10 2 6	10 2 6 10 2 6	10 2 6 10 2 6	10 2 6 10 2 6	10 2 6 10 2 6	10 2 6 10 2 6	10 2 6 10 2 6

Temperature: 106° 105° 104° 103° 102° 101° 100° 99° (Normal Temperature) 98° 97°

Pulse.	128/136	118/124	112/124	132/128	116/112	134/120	112/106
Respir	20/20	20/22	16/18	16/18	16/14	20/16	16/18
Bowels.							
URINE. Amount.		XXVI		XXX	XXVIII	XXVII	XXXII
Sp. Gr.							
Albumen	H Trace	T	H.T	H.T	H.T	H.T	H.T
BLOOD				Trace.	Trace.	Trace	Trace.
Casts	Hyaline				.1	.32	.2

Remarks: Lavage albuminuria


Sept.
Oct.
Disease—
Name—
Arthur Arnold
Age—
12 yrs.
Admitted—
Discharged—
Onset—
Date
Day of Disease
Time
106°
105°
104°
103°
102°
101°
100°
99°
Normal Temperature
98°
97°
Pulse.
Respir
Bowels.
URINE.
Amount.
Sp. Gr.
Albumen
BLOOD
Casts


December 1906
Disease—
Date
Day of Disease
Time
Name—
Arthur Arnold.
Age—
18 yrs.
Admitted—
Discharged—
Onset—
106°
105°
104°
103°
102°
101°
100°
99°
Normal Temperature
98°
97°
5
16
17
18
19
20
21
Pulse.
Respir
Bowels.
URINE.
Amount.
Sp. Gr.
Albumen
BLOOD
Casts
FT
nil
nil
nil

quantity of blood and albumen, from 30 to 35 ounces passed in 24 hours. The cervical adenitis remains hard and brawny, but not particularly painful.
April 25th. Most of the deposit has disappeared, the ulcers are healing. Condition of the patient much better. Temperature coming down. The papules over the joints have almost gone.

After this date the patient improved rapidly. Blood was still present in the urine in small quantities, until the middle of May. The patient was discharged on June 11th, a very faint trace of albumen persisting.

No cardiac changes were at any time noted. The diet ordered was milk, thin custard, and beef juice.

At the early stage, when joint pains were complained of, 15 grains of Salicylate of soda was ordered every four hours. Locally the mouth and throat was treated by frequent swabbing with Izal, and glycerinum boracis. Later a mixture containing iron was given.

III. A. A. Male. Age 18 years.

First fell ill on September 11th 1906. The usual initial symptoms were present, and the rash appeared on September 12th. He was admitted to hospital on September 15th with an attack of moderately/

moderately severe scarlet fever.

September 16th. Complains of pain, on breathing deeply, in the left side. Examination of the heart and lungs reveals nothing.

September 18th. Pain complained of in the right side, examination is negative.

Salicylate of soda appeared to relieve the pain complained of. The temperature fell by lysis, and reached the normal on September 21st, the 11th day of disease. The patient was allowed cut of bed on September 28th and transferred to a convalescent ward. On October 1st; he was apparently doing well.

October 3rd. (23rd day of disease). Temperature 103.8°. Very tender adenitis right side of the neck. Sore throat is complained of, the tonsils are much swollen and congested. Marked general desquamation. Trace of albumen in the urine.

October 4th. Throat better, no complaint.

October 5th. Temperature 104°. Glands of the neck are painful and enlarged again.

October 6th. Temperature still keeping up. Adenitis very marked. Swelling, injection, and tenderness of the gums; there is slight ulceration close to the teeth behind the upper incisors. Complains of pains in the knees, nothing on examination.

Examination/

Examination of heart reveals nothing.

October 7th. Temperature 104.6°F. at 2 a.m. Slept at intervals. Very marked swelling of the tonsils, there is a continuous sheet, of thick, firm deposit on each.

The gums are still very much swollen, there are some haemorrhages into their tissues especially at the roots of the upper incisors. The breath is very offensive. The tongue is furred.

A papular eruption has appeared on the buttocks, the papules are numerous and mostly about the size of a large pin-head, a few have haemorrhages into their tissues. On the right elbow there is one large papule about the size of a pea, it has a haemorrhagic summit; some papules are also present over the left olecranon, but they are not haemorrhagic. There are a few simple papules on the front of the knees. Adenitis is still very marked. No pain is complained of in the limbs. The heart sounds are normal.

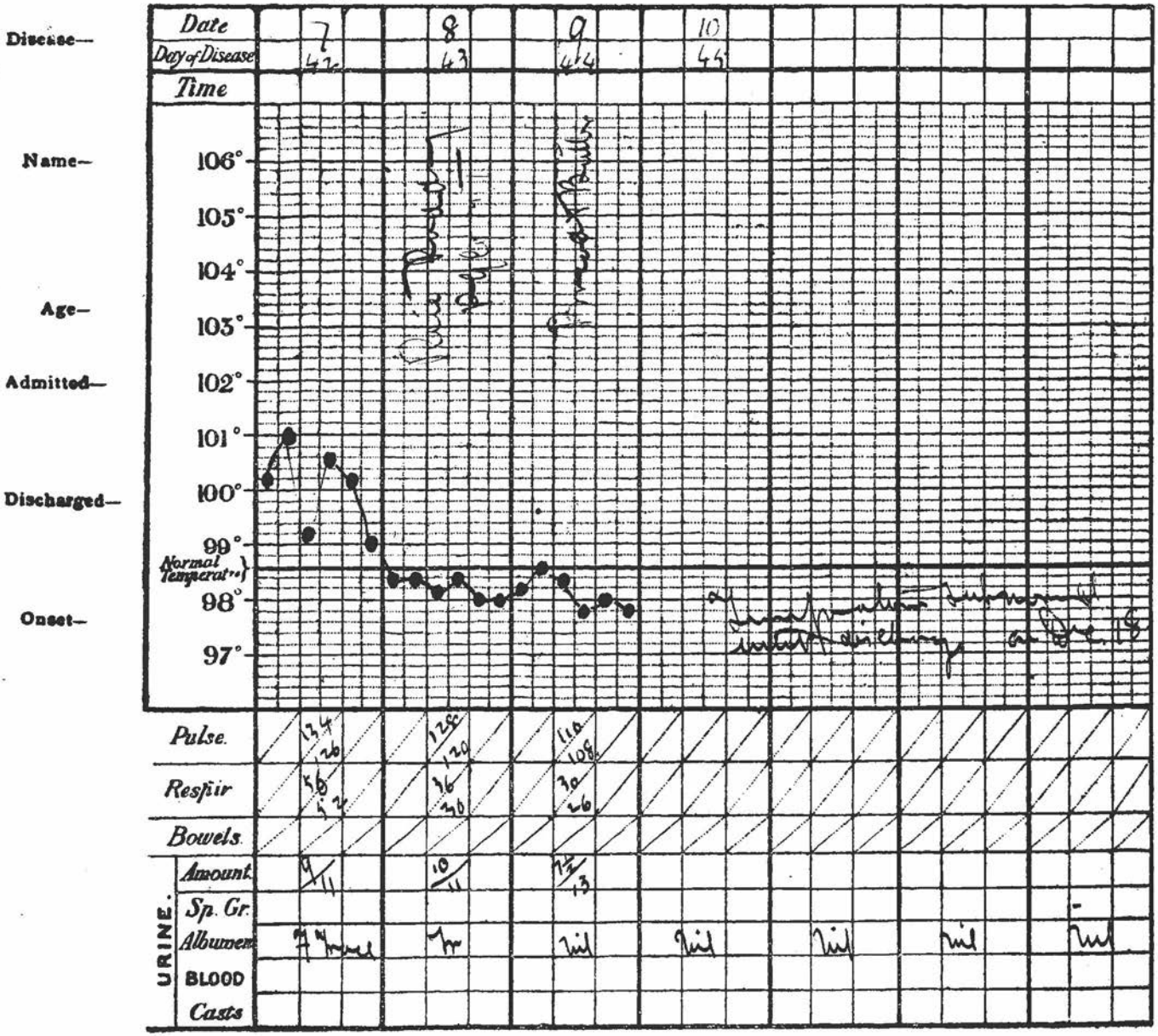
October 8th. Very restless at night. Necrosis of the gums is spreading. The tissues of the gums behind the upper and lower incisors are black and are falling away from the teeth. The spots on the left elbow are now haemorrhagic, they are not so large as those on the right. The condition of the fauces/

fauces is much the same as yesterday, swelling is very marked, there is a thick coating of deposit. Foetor is very marked.

October 9th. Again a very restless night. Slept only at short intervals. The swelling of the faucial structures caused such difficulty in breathing and alarmed the patient so much that it was thought right to prepare for tracheotomy, there were however no actual laryngeal symptoms. Fluids can only be swallowed with some difficulty. There is profuse, thin, sanguineous discharge from the nose. Ulceration of the gums spreading backwards along the teeth. Urine, which for several days has contained a heavy trace of albumen, now has a quantity of blood.

9 o'clock p.m. Subcutaneous haemorrhage about the size of a florin, over the right scapula. Patient appears to be resting better to-night, not the same difficulty in breathing.

October 10th. Slept much better, taking nourishment more freely. Breath not so offensive.
Deposit is clearing off the tonsils, leaving extensive superficial ulceration.
Ulceration has not extended since yesterday. Sloughs as separating from behind the upper incisors, the teeth are all firm. The haemorrhagic spots are drying/


Disease—
Name—
Age—
Admitted—
Discharged—
Onset—
Date
Day of Disease
Time
7
8
9
10
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
Normal Temperature
Pulse.
Respir
Bowels.
URINE.
Amount.
Sp. Gr.
Albumen
BLOOD
Casts

Disease—

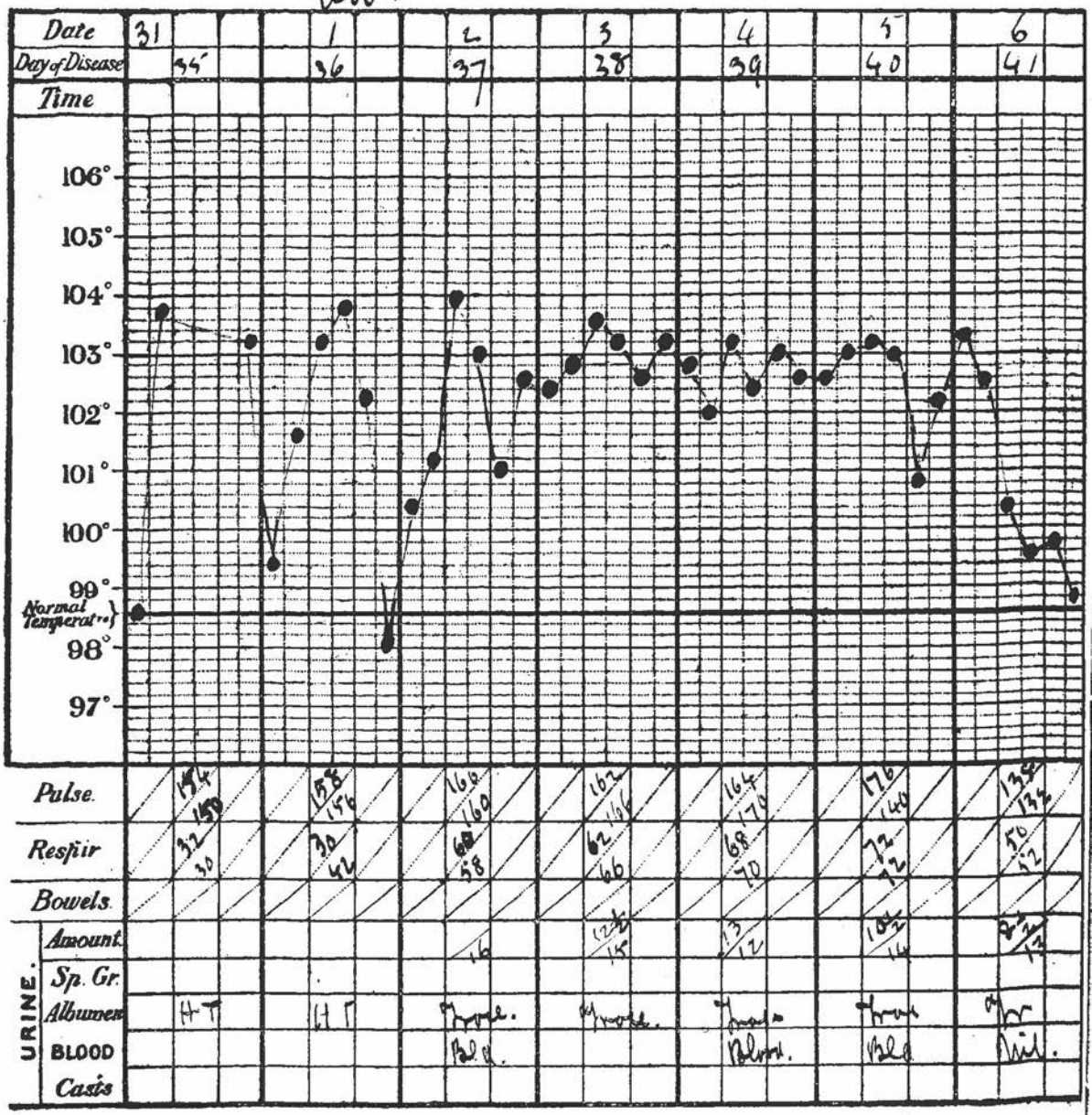
Name—

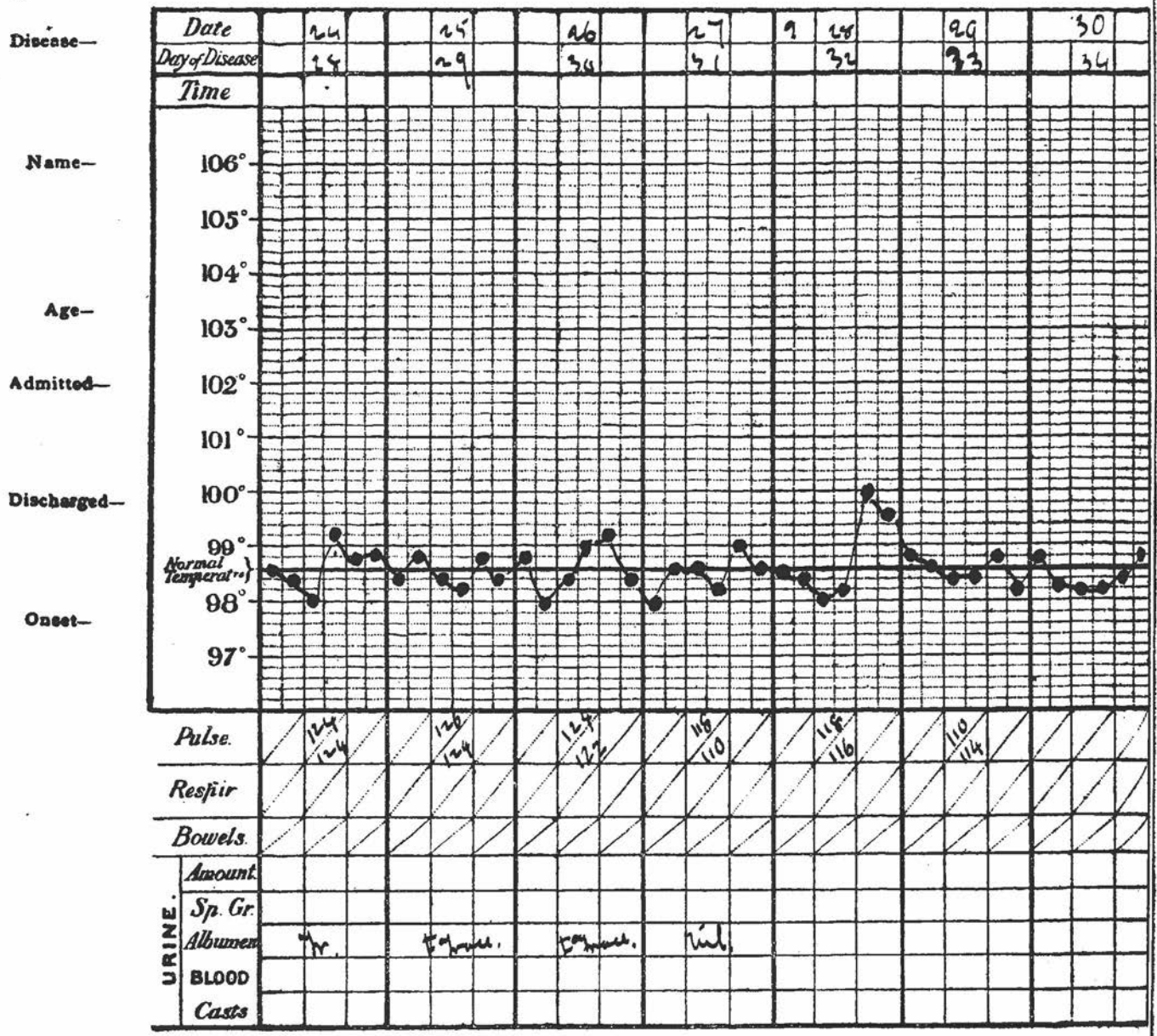
Age—

Admitted—

Discharged—

Onset—




Disease—
Name—
Age—
Admitted—
Discharged—
Onset—
Date
Day of Disease
Time
24
25
26
27
28
29
30
24
29
34
31
32
33
34
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
Normal Temperature
Pulse.
124 124
126 124
124 122
116 110
116 116
110 114
Respir
Bowels.
URINE.
Amount
Sp. Gr.
Albumen
BLOOD
Casts
Trace.
Trace.
Trace.

Disease—

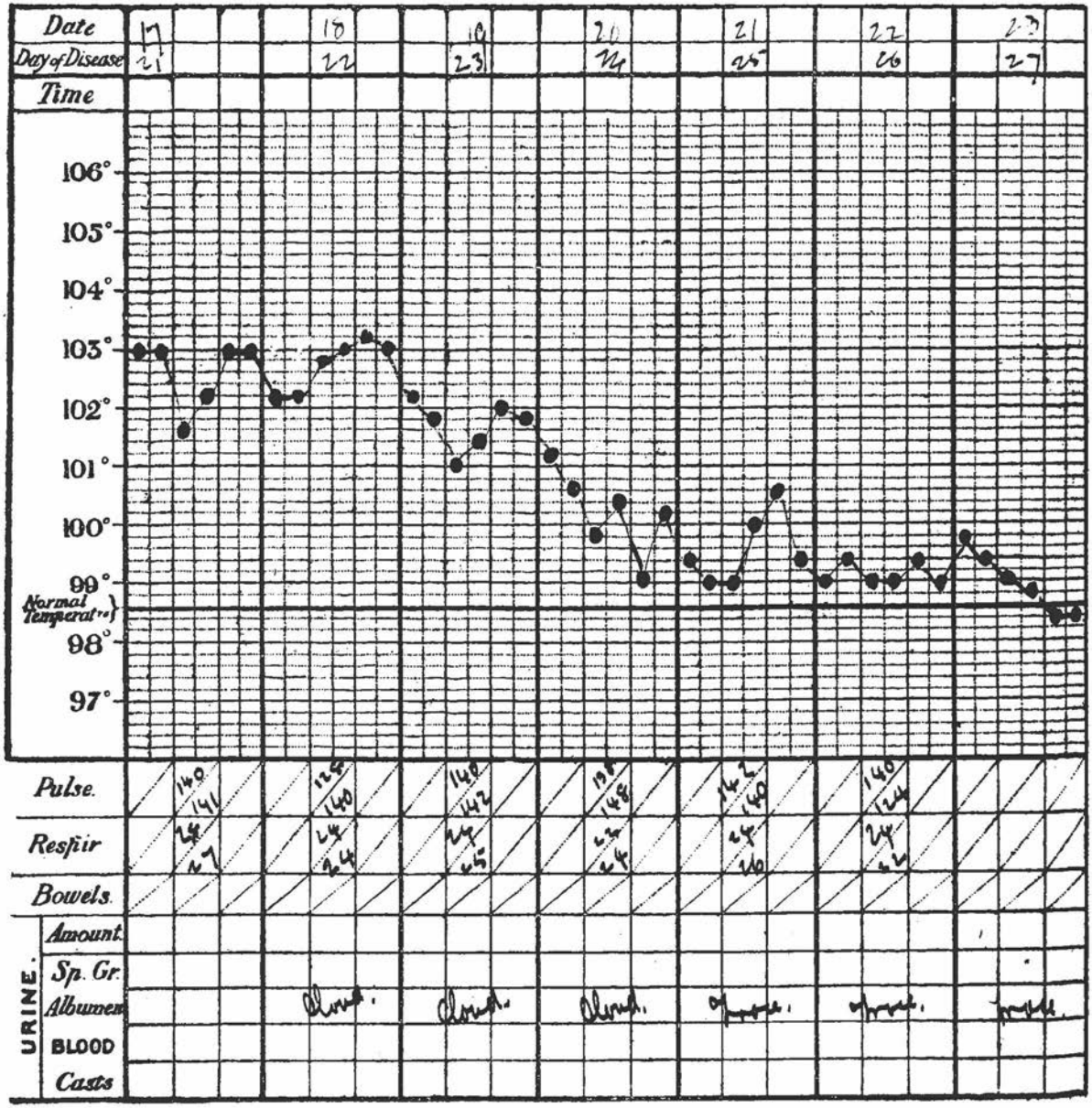
Name—

Age—

Admitted—

Discharged—

Onset—



Disease—

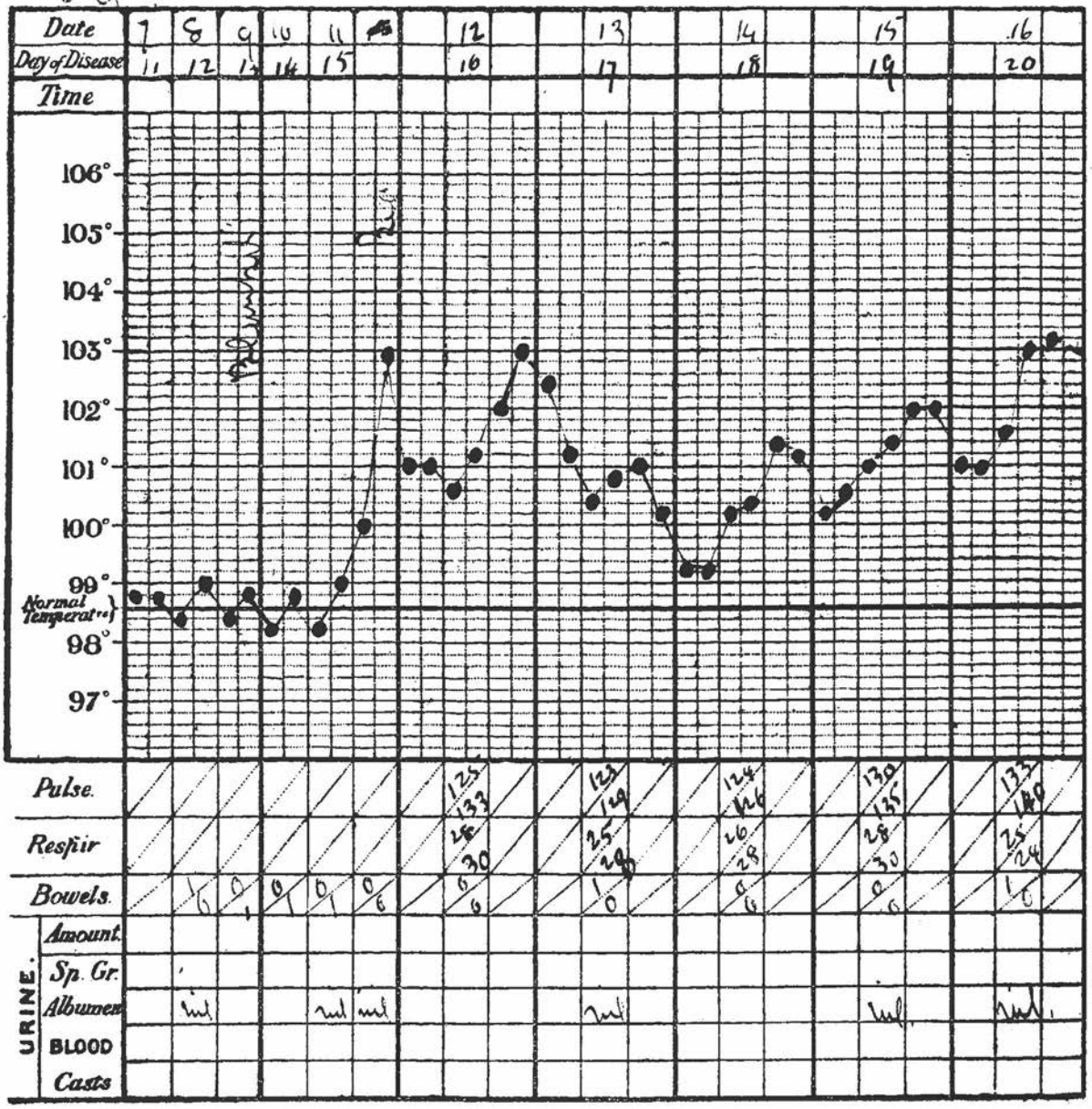
Name—

Age—

Admitted—

Discharged—

Onset—



Sept. Oct.

Disease—

1502.

Name—

Age—

Admitted—

Discharged—

Onset—

Ill.
Bed.
Headache
thirst
vomit
rash &c.
measles.
2-7

Date	30			1			2			3			4			5			6		
Day of Disease	4			5			6			7			8			9			10		
Time																					
106°																					
105°																					
104°																					
103°																					
102°																					
101°																					
100°																					
99°																					
Normal Temperature																					
98°																					
97°																					
Pulse.																					
Respir																					
Bowels.				0	1		0	1		0	1		0	0		0	1		0	1	
URINE. Amount.																					
Sp. Gr.																					
Albumen				nil			nil			nil						nil					
BLOOD																					
Casts																					

drying up, and forming scabs. Heart sounds are normal. Blood and albumen are still present in the urine, .1% of albumen. The deposit is heavy, and consists of blood corpuscles, with some blood and epithelial casts.

From this time onwards the progress of the patient was satisfactory, except that suppuration followed in the glands of the neck. The haematuria and albuminuria gradually passed away.

Treatment adopted was, frequent swabbing with Izal lotion, 12 grains of calcium chloride were given every four hours, beginning on October 7th.

Cultivation of the throat at the height of the attack, shewed staphylococci and streptococci.

IV. P.L. Male. aged 6 years.

Was admitted to hospital on the 4th day of disease suffering from a moderate attack of undoubted scarlet fever. The fauces were markedly injected, there was no exudation, the tongue was typical, and there was a well marked general punctiform rash.
Date of admission, September 30th 1905.
October 1st. Heart and lungs are normal.

The progress of the case was that of a typical scarletina. No further notes were taken until October 11th the 15th day of disease.
October 11th/

October 11th. Moderate cervical adenitis on both sides. Ulceration of the lips. Temperature 101°F.
October 13th. Adenitis is more marked and painful. The tonsils are perhaps somewhat injected and swollen, there is some flaky deposit. Cultivation on blood serum shewed streptococci and a few staphylococci. Temperature 101°.
October 15th. There is a marked swelling and induration of the tissues of the upper lip, on the left side, in the region of the ulcerated part. Complains of pain in the mouth, there is much swelling and injection of the gums, with a great tendency to bleed. Ulceration of the gums close to the upper and lower molars. The breath is very offensive.
October 16th. The breath is more offensive than yesterday, one can only examine the patient's mouth after first plugging one's nostrils. On the left side of the upper jaw, there is necrosis of the tissues of the gums as far forward as the canine on that side. Below, there is extensive necrosis on the inner side of the gums, extending almost all the way round. Some of the teeth are loose, but all are present. The swelling of the lip has extended to the left cheek. Complaining of pain in the region of the wrist/

the wrist joints, nothing noticeable on inspection.
October 17th. Slept badly, not taking nourishment well. Temperature 103°. Gangrene in the mouth has spread forwards towards the middle line. Papules have appeared on the legs, they are most numerous on the extensor aspects; each papule has a haemorrhage into its tissues, they vary in size from a pin-head to a pea; a few are also present on the extensor aspects of the hands and arms. Patient still complains of acute pains in the wrist joints, and is very fretful and irritable.
October 18th. Restless at night. Foetor particularly offensive. Condition of the mouth much the same, except that the gangrene appears to be still spreading. Profuse, offensive, blood-stained discharge from the mouth and nose. Throat again cultivated, with the same results. The urine on boiling throws down a heavy cloud of albumen. There is no haematuria.
October 19th. Condition certainly no better. Patient looks very ill, the pulse is very feeble. There has just been a sharp attack of bleeding from the mouth, no definite bleeding point is to be found. There is a haemorrhagic discharge from the left ear.
October 20th. Temperature falling. Has had a better night. Taking food well. Condition of the mouth much/

much the same, sloughs however, appear to be separating. Many of the teeth are loose, but all are present. Papules on the limbs have lost their haemorrhagic character, and are now pustular. There is marked swelling and induration round the sore on the lip, but the gangrene does not appear to be extending into its tissues.

October 21st. Improvement continues. Gangrene certainly not extending, sloughs separating. Foetor is not nearly so marked.

October 24th. Sloughs have all separated, and offensive odour has gone. The front lower teeth are bared of gum and loose.

October 28th. Four lower incisors removed, the alveolar margin in the region is denuded of gum tissue and periosteum. The canines and premolars below are loose, the bone surrounding them is denuded of soft tissues. Upper teeth are all quite firm.

The temperature which reached the normal on October 21st remained so until October 31st. The condition of the mouth continued to improve, and the patient was apparently doing well. A faint trace of albumen remained in the urine.

October 31st. Temperature 103.8°F. Not complaining but fretful. Nothing found on examining the chest.

November 1st./

November 1st. Said to have "vomited" two ounces of blood in the night; probably however it came from the mouth, as there is a blood-clot between two upper molars. Respirations are hurried, cough suppressed. No definite signs of consolidation of the lungs.

Later. One or two ounces of blood from the mouth.

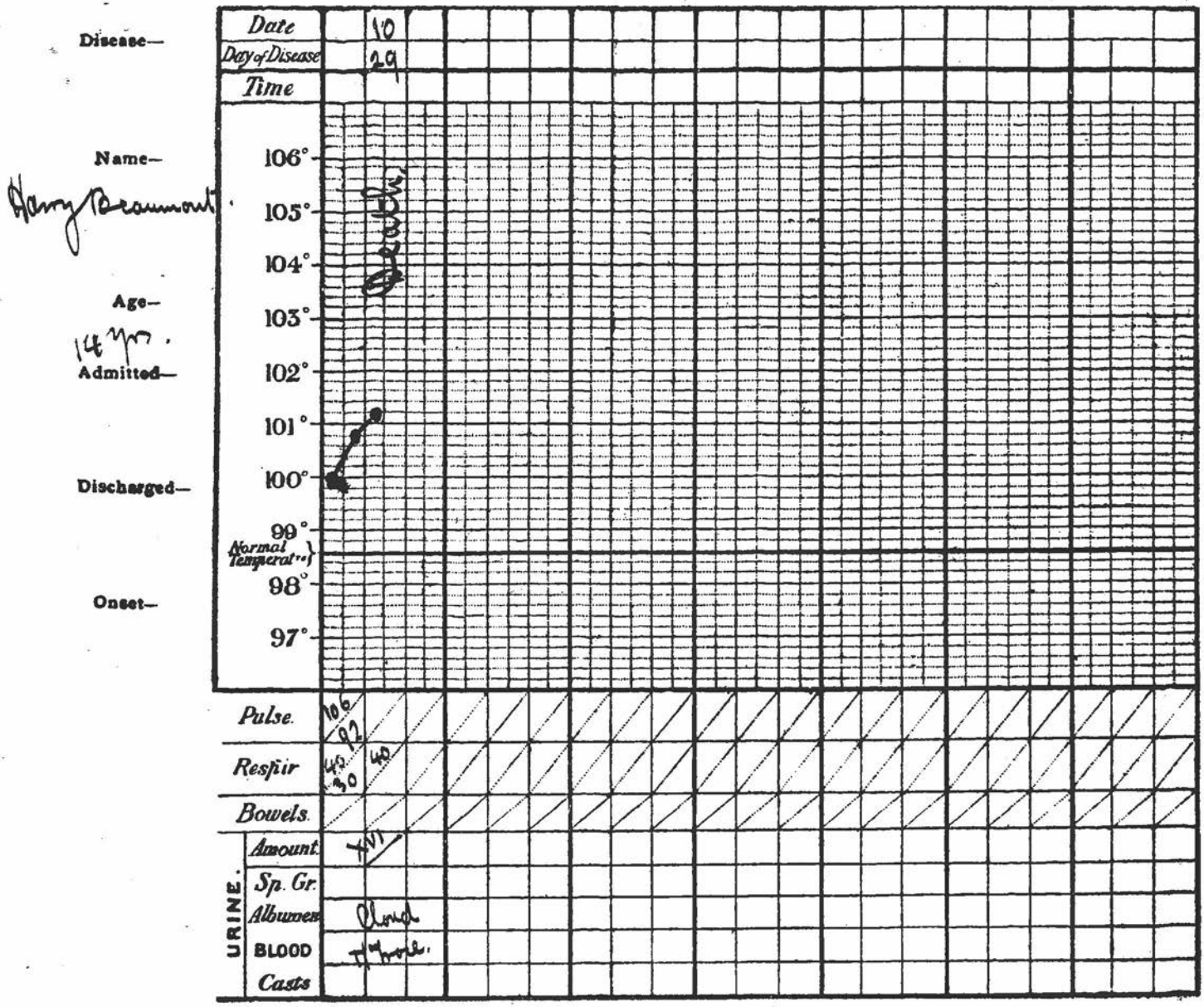
November 2nd. Temperature running about 103°. Cloud of albumen, and moderate trace of blood in the urine. Indefinite signs of pneumonia over the right scapula. Examination of the abdomen reveals nothing. Heart very rapid, with well marked, blowing, systolic, murmur, conducted into the axilla. There is no swelling of the joints, and no signs of mastoid trouble. No rigors.

November 3rd. Pneumonia over the right scapula quite definite. Urine as yesterday.

November 4th. Physical signs unchanged. Temperature keeping steady at 103°F.

November 6th. Blood has disappeared from the urine, still trace of albumen. Physical signs are now well marked in the right axilla, and at the apex, in front and posteriorly.

November 7th. Temperature is down. Patient quite comfortable. Sequestrum has separated from the lower jaw, it consists of the bony framework that had surrounded/


Disease—
Name—
Harry Beaumont
Age—
14 yrs.
Admitted—
Discharged—
Onset—
Date
Day of Disease
10
29
Time
106°
105°
104°
103°
102°
101°
100°
99°
Normal Temperature
98°
97°
Pulse.
106
92
Respir
40
30
40
Bowels.
XVI
URINE.
Amount.
Sp. Gr.
Albumen
Clear
BLOOD
Trace.
Casts

May.

Disease—

Name—

Wm Beaumont.

Age—

14 yrs.

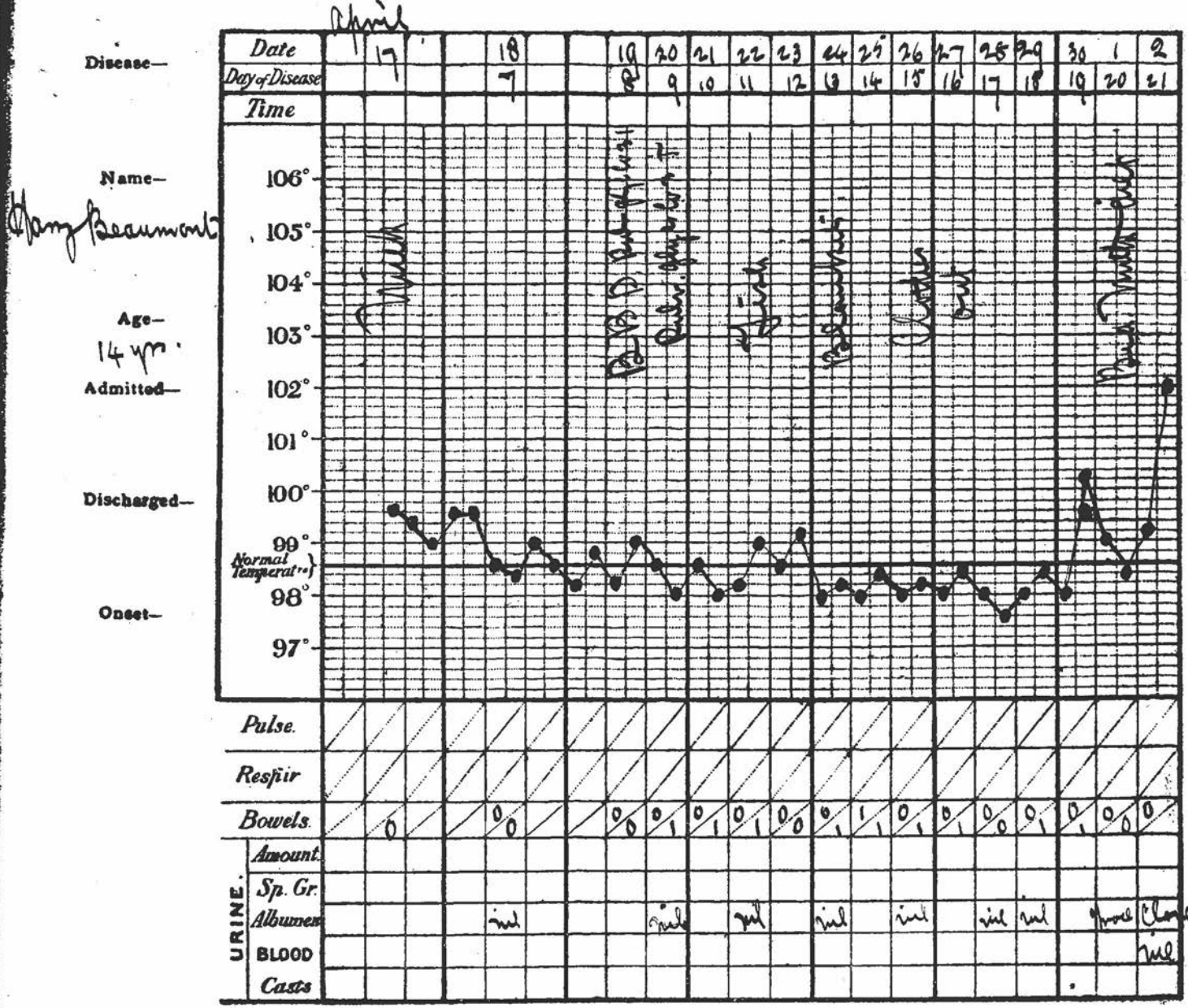
Admitted—

Discharged—

Onset—

Date	3	4	5	6	7	8	9
Day of Disease	22	23	24	25	26	27	28.
Time							
Pulse.				108/112	108/116	104/112	106/120
Respir				28/36	36/36	32/30	34/32
Bowels.	1/0	1/1	0/0	1	1	3	1/0
URINE. Amount.			XXV/XV	XXIII/XIV	X/XIV	XIV/VIII	VIII/IV
Sp. Gr.							
Albumen	Cloud.	Cloud.	Cloud.	Cloud.	Cloud.	Cloud.	Cloud.
BLOOD	no	no	no	no	Trace	Trace	Trace.
Casts							

106° 105° 104° 103° 102° 101° 100° 99° Normal Temperature 98° 97°


April
Disease—
Date
Day of Disease
Time
Name—
Harry Beaumont
Age—
14 yrs.
Admitted—
Discharged—
Onset—
106°
105°
104°
103°
102°
101°
100°
99°
Normal Temperature
98°
97°
Pulse.
Respir.
Bowels.
URINE.
Amount.
Sp. Gr.
Albumen
BLOOD
Casts

surrounded the lower incisors.

From this onwards the patient improved rapidly, the albumen disappeared from the urine on November 9th, and he was discharged quite well on December 18th.

The local treatment employed was Izal and glycerinum boracis for the mouth. Salicylate of soda was given during the early stages, when joint pains were complained of; later chlorate of potash and iron were ordered.

Stimulants were freely used, during, both the attack of tonsillitis with stomatitis, and the pneumonia with nephritis which followed.

V. H. B. Male aged 14 years.

First fell ill on April 12th 1905. Admitted to Hospital on April 17th, with typical mild attack of scarlet fever. Uneventful recovery from initial symptoms.

May 2nd. Temperature 102°. Cloud of albumen on boiling the urine.

May 3rd. (22nd day of disease). Albuminuria being treated with blanket pack, skin acting well. Complaining of sore throat, considerable swelling and injection of fauces generally, there is some firm white deposit with definite edges on both tonsils. Cultivation shews streptococci and staphylococci. Moderate/

Moderate double cervical adenitis. Heart sounds are clear, no murmur.

May 5th. Condition of patient remains much the same. The faucial inflammation is perhaps more marked. The breath is somewhat offensive. The gums are injected and spongy.

May 6th, evening. The temperature has gone up to 104°. The tonsils are very markedly swollen, there is a thick, firm, white deposit on the tonsils, which forms a continuous sheet on each side. The tonsils almost meet in the middle line. Cultivation give an almost pure culture of streptococci.

May 7th. Heavy trace of blood in the urine. Adenitis moderate. Fauces much as before. Complains of pain in the region of the praecordia; a soft systolic murmur is heard all over the praecordia, loudest between the left nipple and the sternum, it is not conducted to the axilla. There is profuse, thin, sanguineous nasal discharge, the odour is very offensive. Patient is in evident distress.

Evening. Very restless. Complaining bitterly of sore throat.

May 8th. 10 a.m. Complaining of acute pain in the left side of the chest, in the mammary and subscapular regions; physical examination reveals nothing. Respirations/

Respirations are hurried and evidently painful. Cough, occasional and suppressed. Fauces are much the same as yesterday. The tongue is heavily coated with a thick white furr. Heart sounds are rapid, systolic murmur is unchanged, the area of cardiac dulness is not increased. Abdomen is somewhat distended generally. Some adbominal uneasiness is complained of, but not localised. Abdominal movement is free, the liver dulness is absolute, the percussion note in the flanks is clear.

1.30 p.m. Pain in the left side of chest is very acute. Cultivation repeated at this time gives the same result as before.

May 9th. Slept very little, Complains constantly of pain in the left side, Tongue is dry and covered with blood-stained crusts. Exudation is still present on the tonsils, but is disintegrating. There is a profuse sanguineous discharge from the nose. The breath is very offensive. Percussion note at the left base is markedly impaired, breath sounds are faint, vocal resonance is diminished. At the right base the breath sounds are masked by moist sounds.

2.30 p.m. Patient very much worse. Considerable cyanosis. Cutaneous veins of the thorax and neck are distended. Percussion note over the left base is/

is absolutely dull. The pulse is pretty good considering the respiratory distress. There is a subcutaneous haemorrhage about the size of a shilling on the calf of the right leg.

Paracentesis thoracis in the posterior axillary line, opposite the inferior angle of the scapula. About twenty ounces straw-coloured, slightly turbid serum was obtained, and some relief to the distress resulted. Result of the bacteriological examination was negative.

10.30 p.m. Patient has slept at intervals through the day, pulse continues good, cyanosis not so marked. Some epistaxis this evening, not alarming.

May 10th. Restless night. Pulse feeble, almost imperceptible at the wrist. Refuses all food. Tongue dry and covered with blood-stained crusts. Nasal discharge profuse and blood-stained, the odour is very offensive. Patient died at 12 noon.

Treatment. The mouth and throat were swabbed frequently with antiseptics. At the onset of the acute pain in the side a hypodermic dose of $\frac{1}{8}$ grain of morphia was administered, this was repeated in two hours. Later, brandy and strychnine hypodermically were given.

I unfortunately was unable to persuade the relatives to sanction an autopsy.

December

Disease—

Name— M. W.

Age—

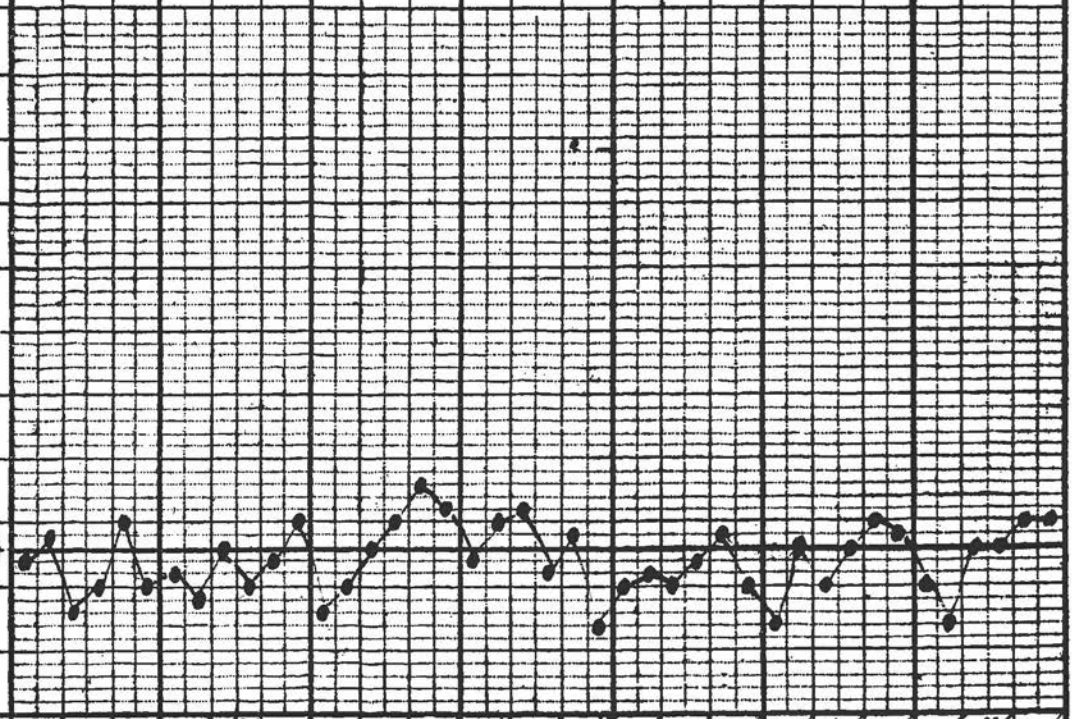
Admitted—

Discharged—

Onset—

Date	13	14	15	16	17	18	19
Day of Disease	41	42	43	44	45	46	47
Time							

106° 105° 104° 103° 102° 101° 100° 99° Normal Temperature 98° 97°



Pulse.	128 / 100	128 / 128	132 / 124	130 / 132	126 / 130	126 / 130	124 / 130
Respir	40 / 40	40 / 38	38 / 36	32 / 30	30 / 30	28 / 26	26 / 24
Bowels.							
URINE. Amount.	XV / X	XVIII / XV	XX / X	XIV / VIII	XII / X	XI / XII	XVII / X
Sp. Gr.							
Albumen	Trace	Trace	Trace	Trace	Trace	Trace	Trace
BLOOD	Trace	Trace and Trace	Trace	Trace	Trace and Trace	Trace and Trace	Trace and Trace
Casts							

December.

Disease—

Name— Mabel Williams

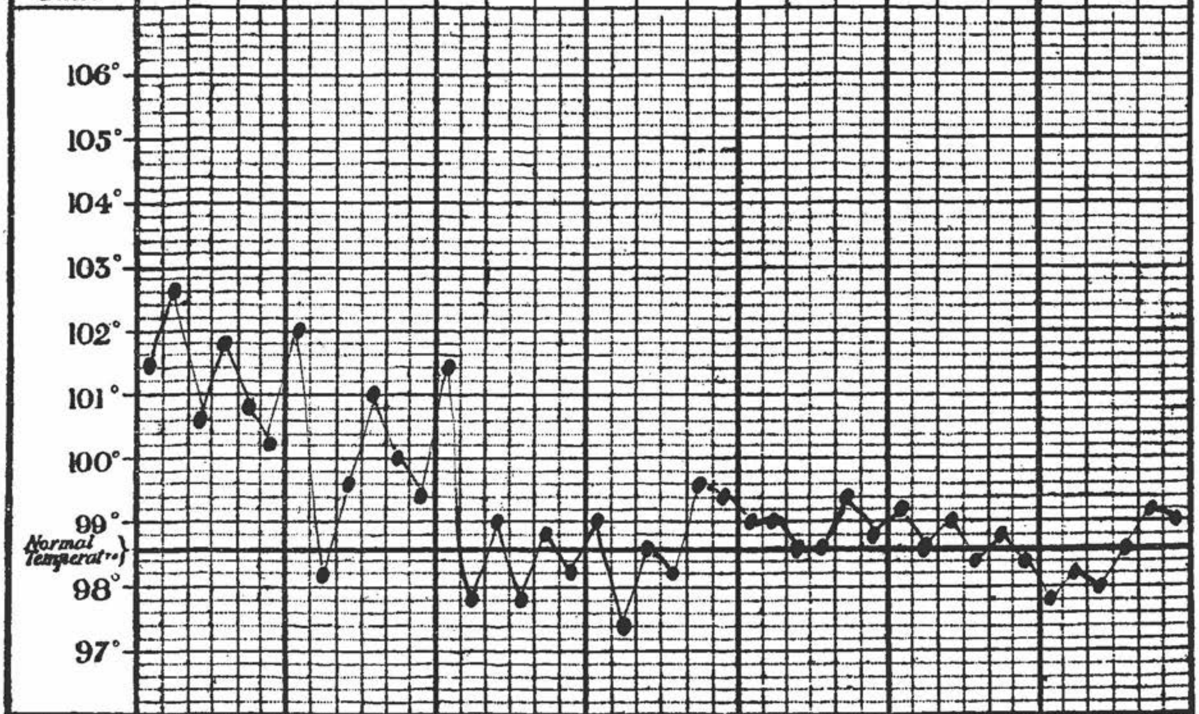
Age—

Admitted—

Discharged—

Onset—

Date	6	7	8	9	10	11	12
Day of Disease	34	35	36	37	38	39	40
Time							



Pulse.	165 / 168	160 / 164	162 / 158	164 / 152	140 / 140	136 / 132	134 / 132
Respir	60 / 56	60 / 62	64 / 68	58 / 54	64 / 60	60 / 56	50 / 48
Bowels.	0 / 0	0 / 1	2 / 1	2 / 1	1 / 1	1 / 0	0 / 1
URINE. Amount.	VIII / XVI	XII / XVII	XIX / X	XV / X	VI / X	X / XII	VIII / XII
URINE. Sp. Gr.							
URINE. Albumen	trace	trace	trace	trace	trace	trace	trace
URINE. BLOOD							
URINE. Casts							

December.

Disease—

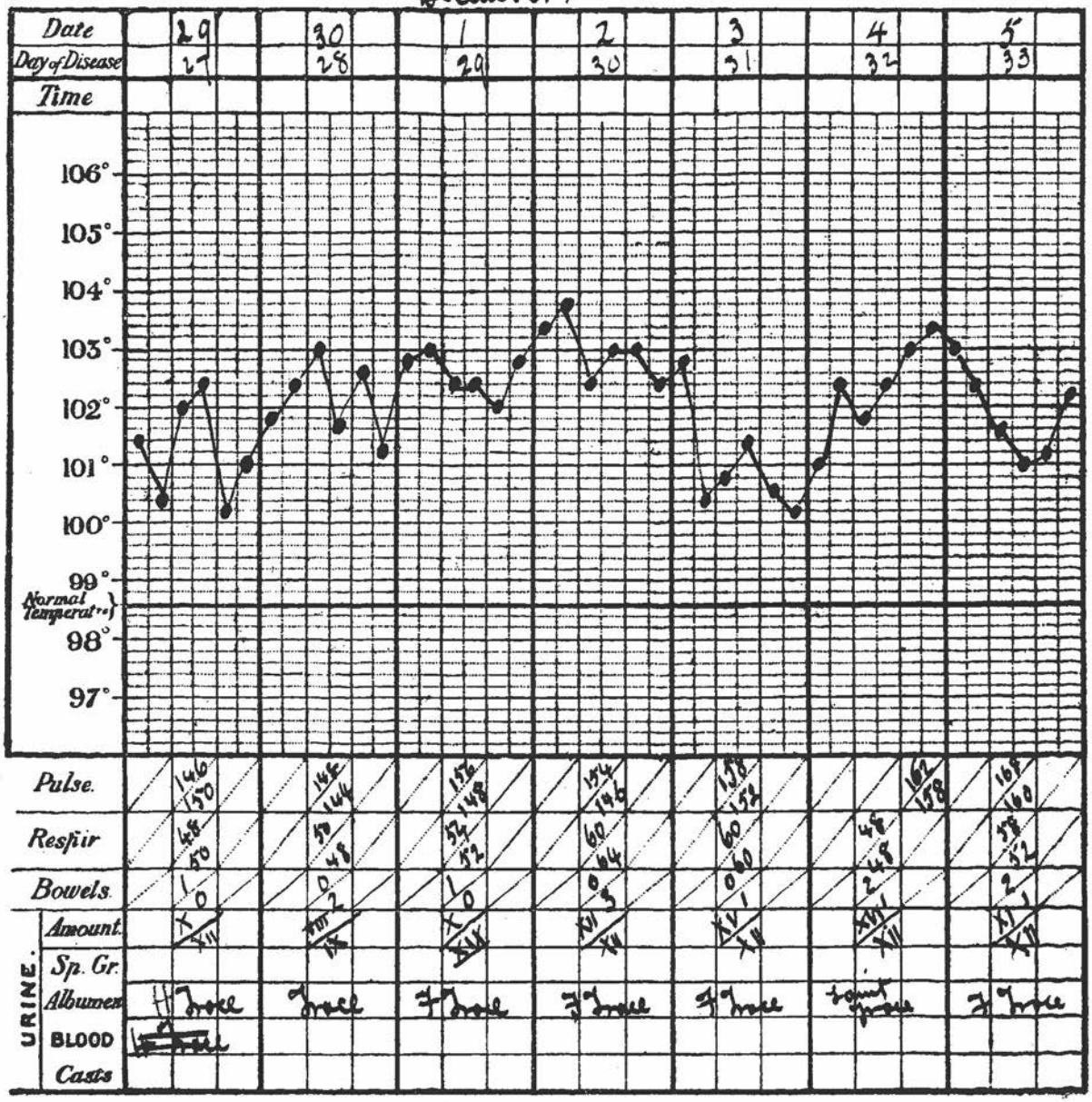
Name— Mabel Williams.

Age—

Admitted—

Discharged—

Onset—



Disease—

Name—

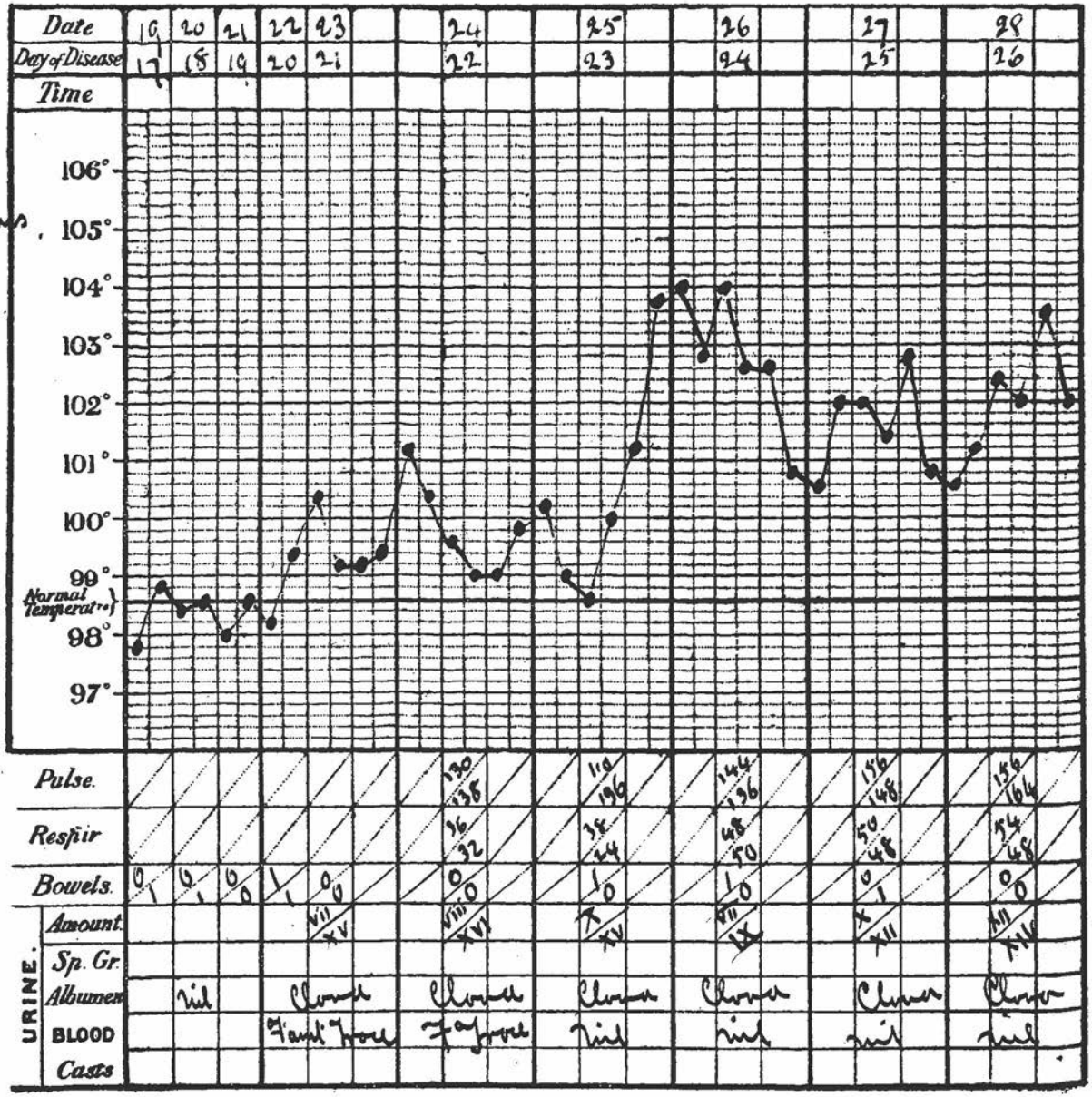
Mabel Williams

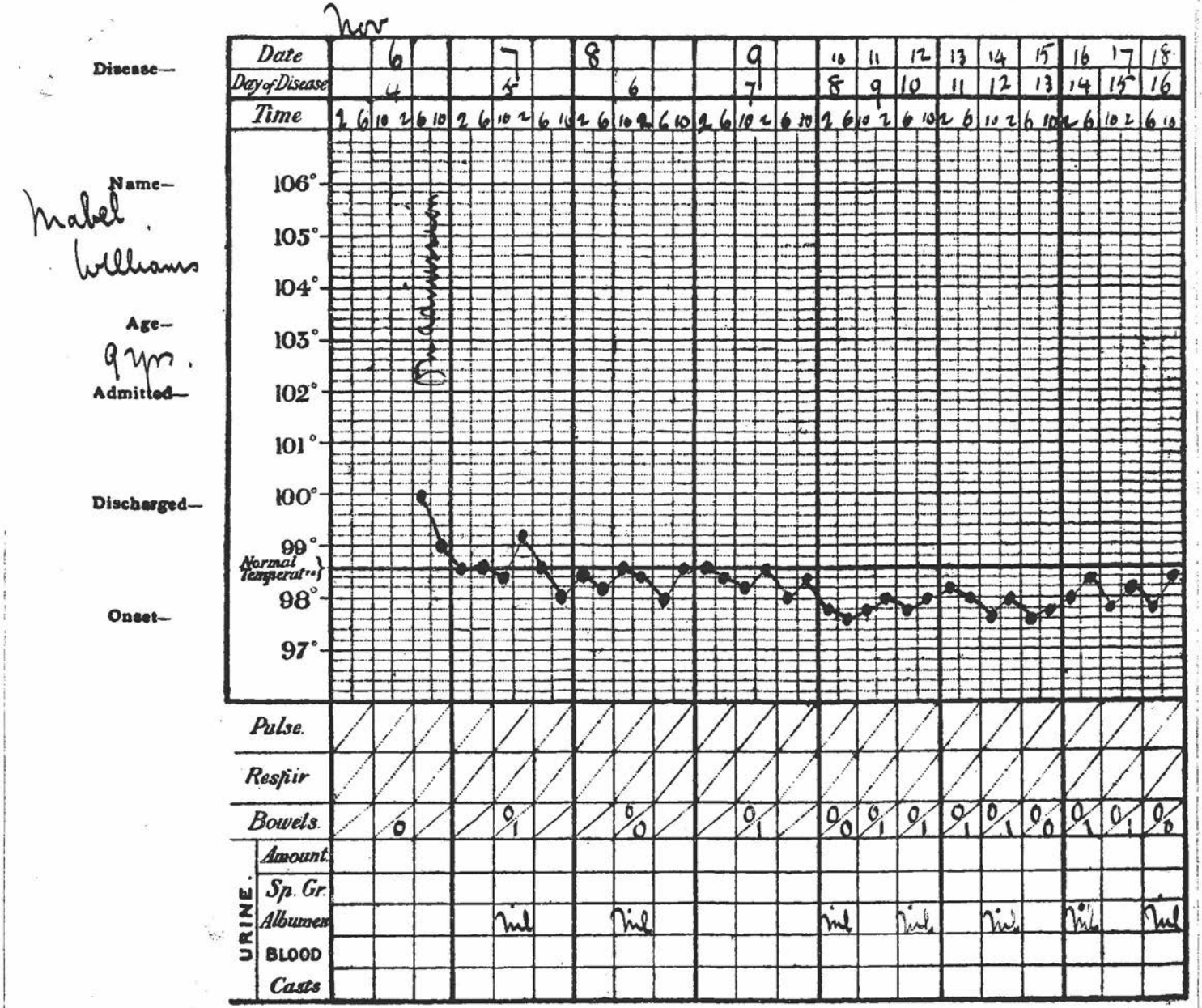
Age—

Admitted—

Discharged—

Onset—




Nov
Disease—
Date
Day of Disease
Time
Name—
Mabel Williams
Age—
9 yrs.
Admitted—
Discharged—
Onset—
106°
105°
104°
103°
102°
101°
100°
99°
Normal temperature
98°
97°
Pulse.
Respir.
Bowels.
URINE.
Amount.
Sp. Gr.
Albumen
BLOOD
Casts

VI. M. W. Female. Aged 9 years.

First fell ill on November 3rd, 1905, and was admitted to Hospital on November 6th.

Patient passed through a definite mild attack of the disease.

November 16th. Patient transferred to convalescent ward.

November 22nd, evening. (20th day of disease) Complaining of sore throat; fauces are somewhat injected, there is no deposit.

November 23rd. Moderate cervical adenitis, left side. Slight flaky deposit on the tonsils. Cultivation on blood serum gives a growth of cocci, numerous streptococci. There is a cloud of albumen in the urine and a faint trace of blood.

November 24th. Fauces are unchanged. Breath is offensive. The gums are injected, swollen and bleed easily, there is no appearance of necrosis.

November 25th. Condition of the patient worse. Temperature, 103.8°F. Pulse is very rapid, feeble and irregular. Cloud of albumen in the urine.

November 26th. Restless night. Respirations very rapid. Distinct impairment of percussion note at the left base; moist sounds, fine and medium, general in both lungs. Fauces cleaning. Adenitis not so marked/

marked. Temperature 104°F.

November 27th. Slept fairly well, taking food well. Improvement in faucial condition continues. Marked dulness, tubular breathing, with fine crepitations and increased vocal resonance at the left base, complains of pain in this region.

November 28th. Temperature 102.4°F. Physical signs much as yesterday, but heard over a much wider area, quite as high as the spine of the scapula.

November 29th. All the left lung is now consolidated, cough is frequent but not painful. Pulse continues fairly satisfactory. There is still a quantity of albumen in the urine, but no blood. The urine is scanty and high coloured with a deposit of urates.

December 2nd. Dulness at the left base still very marked, breath sounds tubular, but not so audible as a day or two ago. Exploring needle introduced with no result.

December 3rd. Dulness over the whole left chest, front and back, numerous moist sounds on inspiration. Pulse 140, moderate force and volume.

December 4th. Restless night. Pulse very rapid and feeble. Some cyanosis. Patch of tubular breathing over the right scapula, no adventitious sounds. At the left base dulness is not so marked, breath sounds are harsh vesicular. Cough is very troublesome.

December 5th./

December 5th. Condition of the patient seems better. Physical signs are unchanged.

December 6th. Soft, short, systolic murmur at the apex. There are friction sounds of a creaking character, synchronous with respiration, below and to the left of the apex beat.

December 8th. Temperature has dropped by crisis. Patient's condition is very much better, but the pulse is still very feeble and rapid.

December 10th. Physical signs clearing up rapidly, except at the left apex, where the dulness and high-pitched bronchial breathing still persist. A heavy trace of albumen is still present in the urine. Patient vomited twice in the night.

December 13th. There is a trace of blood in the urine to-day, no complaint, no oedema, passing sufficient urine. The deposit contains some red cells, and some blood and epithelial casts.

The haematuria and albuminuria slowly disappeared. Physical signs at the left apex persisted for some time, and a diagnosis of phthisis was suggested, but no tubercle bacilli were found in the sputum. The patient later put on flesh rapidly, and with the exception of a small axillary abscess from an infected hair follicle, there is nothing else to record.

The patient/

The patient was discharged on February 12th. I saw her six months later, she was in an excellent state of health; with the exception of slight flattening below the clavicle on the left side, there were no signs of either pulmonary or cardiac disease.

The first of these cases that I saw puzzled me very much, I had never read nor heard of any condition that was quite like it, and the question arose, had the affection anything to do with the original attack of scarlet fever?, which in this case was of a particularly mild character. When the other cases occurred I was struck by the remarkable uniformity in the onset, symptoms, and course of each. A search through the standard English text-books, both on general medicine and on fevers, threw no light on the subject. Caiger[1] in Clifford Allbutt's System dismisses the subject of "secondary tonsillitis" in a few lines, he says that it is rarely severe and that the liability to it appears to be equal in all stages of the convalescence.

Von Jurgenson [2] describes a case that has so much in common with those I have seen, that I take the liberty of quoting it rather fully.

The patient, a girl aged 12 years, became ill on March 2nd. 1889. The attack evidently was a moderately/

moderately severe one, and pains in the wrist joints are reported on March 6th. On March 18th, the 17th day of disease, the pains returned again with greater severity, the fingers, wrists and elbows being affected. At the same time there appeared on the extensor surfaces of the forearms, papules with haemorrhagic centres. There was vomiting. The eruption later appeared on the buttocks and on the lobes of the ears. There was albuminuria.
"March 20th. Again there is a fresh membrane formation in the throat, in different areas with a tendency to confluence. The skin involvement on the extensor surfaces of the elbow joints has changed into great haemorrhagic gangrenous patches surrounded by a red inflammatory ares."
"March 22nd. Joint enlargements diminished, fresh eruption on the thighs, fresh haemorrhagic nodules on the upper arms.

In the pharynx an extensive necrotic membrane, discoloured, gangrenous, and fast clinging patches; and intense pallor; the gums swollen, bleeding easily; a marked gangrenous, serous nasal discharge."
A cardiac murmur appeared on March 25th. From the end of March onwards the improvement in the mucous membranes was rapid, and with the exception of erysipelas lasting from April 22nd to April 25th, there is nothing recorded/

recorded.

Dr. J. O. Syme [3] records a number of cases of, what he calls, secondary rashes in scarlet fever; these rashes were either erythematous, urticarial, eczematous, papular, or haemorrhagic. The earliest day on which the symptoms appeared, was the 14th from the onset of the primary attack, but most were much later, from the 18th to the 26th day; in one case no symptoms were noted until the 52nd day. They all exhibited a marked uniformity in symptoms. Adenitis, recrudescence of the early faucial inflammation, albuminuria, rash, and fever, were present in all cases. In three of the ten cases the rash appears to have been similar to that present in mine, that is to say, papular and haemorrhagic. From Dr. Syme's description however, I should not think that his cases were so severe as those in my series, in none of them was there a fatal issue. No mention is made of the age of the patient. It is noted that the initial attack was, in most of them, severe.

Dr. Bowie [4] writing on the leucocytosis of scarlet fever and its complications, discusses the condition known at Monsall Fever Hospital, Manchester, as "secondary throat." "This complication or group of complications" he says, is usually severe, the throat/

throat and nares may be affected alone, but frequently there is also adenitis and nephritis. He found the leucocytosis to reach as high as 30,000 or 40,000.

Dr. Bowie only gives details of two cases, in one the symptoms began on the 18th day of scarlet fever, in the other on the 15th. In both, the initial attack was not a severe one.

Dr. Wm. Hunter (5) describes in detail a case of, what he calls, "secondary angina" in a female patient aged 24, convalescent from a mild attack of scarlet fever. The case ended fatally six days from the onset of the symptoms. Death apparently was due to the intense severity of the tonsillitis, the adenitis, and the cervical cellulitis. There is no history of skin rashes, haematuria, or joint pains. Dr. Hunter mentions the condition in support of his idea, that the secondary complications of scarlet fever are due to oral sepsis; the woman on admission had an alveolar abscess.

Dr. Manning (6) in a paper entitled, "The skin eruptions which occur in the Septicaemia following scarlet fever and Diphtheria," has described a series of cases in which rashes of an erythematous or macular character appear in the third week of the disease/

disease; they were associated with extreme wasting, and sloughing of the tissues of the throat. They occurred in 2.1 per cent of 6,000 cases, and were twice as frequent among patients under 5 years as among all those older. I do not think these cases can have much in common with those under discussion, they appear to me to conform rather to the septic rash, as we call it at Lodge Moor. This is frequently seen in the worst cases of Anginous Scarlet Fever in young children, when the faucial ulceration is extreme, and there is associated adenitis, rhinitis and otorrhoea. The rash usually appears, as a raised, bluish or dark red, macular eruption, on the cheeks, nose, buttocks and extensor surfaces of upper and lower limbs. In the worst cases it may become general, but it is sometimes evanescent, almost disappearing, and then reappearing in a short space of time. I have learnt to look on its occurrence as an event of bad omen, the majority of the cases die. I have the notes of one case where, in a boy aged 8 years, the throat, nose and glandular condition somewhat improved, but the rash remained, the wasting was extreme, and, after running a high temperature for a week or ten days, death occurred. I mention this case in particular because it seemed to me as if the/

the efforts at repair in the fauces and upper air passages were partly successful, but the blood poisoning contracted in the early stages could not be combated.

Dr. Mahomet (7) observed that between the 18th and 22nd day there were certain changes that frequently occurred. These were albuminuria, rise of blood pressure, diminished excretion of urea, and diarrhoea. He does not mention secondary throat nor the occurrence of haemorrhagic rashes.

With these exceptions, I have been unable to find any cases that in any way resembled mine. Had it not been for the fact that I had seen more than one, and that they presented so many features in common, I should have been inclined to consider them some rare form of gangrenous tonsillitis or pharyngitis, associated with general septic intoxication, and perhaps I might have doubted their connection with a mild attack of scarlet fever, the acute symptoms of which had subsided a fortnight or three weeks previously.

I might now discuss the different features of the cases in detail.

The onset in all was the same. The patient complained of pain in the throat, and on examination all the faucial structures were found to be intensely congested/

congested and oedematous. This was followed by swelling of the glands of the neck. The deposit which appeared later had a remarkably firm consistence, so that, although one could not say that it had all the typical characteristics of diphtheric membrane, it was thought best in two of the cases, to inject the antitoxin at once. The extreme swelling, injection, and painfulness of the condition, was all against the diagnosis of diphtheria; in no case was the Klebs Loeffler bacillus present, but streptococci were very noticeably present in all the growths.

Dr. Goodall (8) says that membranous deposit is found in the throat of scarlet fever patients in two forms, during the early angina, and as diphtheric membrane in the convalescent stage. This opens up the question of post-scarletinal diphtheria, a subject I intend discussing later.

The tissues of the gums participated in the inflammatory changes, becoming injected, painful and swollen. In cases I, III, and IV, the inflammation went on to gangrenous ulceration, while in I, and III, it was extreme, resulting in loosening of the teeth and necrosis of the underlying bone. Stomatitis in a severe form is a well recognised complication of/

of scarlet fever, but it appears to be that form which appears in the acute stage of angiose cases, and particularly in young children who strenuously resist all attempts at treatment. In two cases of this type, I have seen gangrene of the entire tissues of the gum, with loosening of the teeth, and necrosis of the jaw. Both cases terminated fatally. Caiger describes the condition, and Henoch (9) discusses it very fully. He has found that "Stomatitis scarletinosa" is usually associated with gangrenous pharyngitis, it begins on the fifth day or later, and may lead to widespread destruction of tissue; he mentions one fatal case in which the jaw was in many places carious and denuded of periosteum. With the exception of that described by Von Jurgenson, I have been unable to find the record of any case in which gangrenous stomatitis set in during the convalescent period.

Pain in the joints was a marked feature in four of my cases, it was also present in those described by Dr. Syme, and in Von Jurgenson's. It appeared very early and caused much distress, but no swelling nor redness was noticed. These pains seem to me to be a condition quite different from the so-called Scarletinal rheumatism. Joint affections do certainly, and not uncommonly, occur in the later stages, but/

but in my experience they are most frequently associated with an acute attack of the disease in adults and older children, and especially in those cases in which the rash is vivid, and the throat symptoms, at the onset, severe.

A tendency to haemorrhage was one of the marked features and was particularly noticeable in Case IV, in which there was free bleeding from the gums. In five of the six cases the appearance of a papular rash, followed by haemorrhage into the tissues of the papules, necessarily arrested the attention; a rash very similar is described by Von Jurgenson and by Dr. Syme. The occurrence of purpuric symptoms at both the early and later stages of the disease I shall discuss later.

Albuminuria and Haematuria were present in all my cases, either appearing at the same time as the throat symptoms or shortly afterwards; taking all the associated symptoms, and the time of onset into consideration, I see no reason why they should not be looked upon as indicative of an acute nephritis. Von Jurgenson, Syme, and Bowie also report nephritis as an accompaniment of the cases they describe.

In the one fatal case, in which an autopsy was performed, the signs of acute inflammation of the kidneys were indefinite; unfortunately it was the first/

first of the series or I might have settled the question by microscopical examination. With regard to this point, it seems to me that, there must still be some work to do on the morbid anatomy of the kidney in the different stages of scarlet fever. In many fatal cases, even quite early in the disease, we find very definite changes in the naked eye appearances, and yet before death little or no evidence of the condition was present. Some writers have remarked on the existence of this latent morbid process in the kidneys, Sörensen (10) is of opinion that more or less inflammatory changes may be present in the kidneys without causing evident or discoverable symptoms, and that the clinical phenomena appear much later than the anatomical changes take place.

I think it is not unreasonable to suppose that the toxins, formed by the secondary inflammatory changes in the throat, and acting on the already damaged kidneys, set up an actual nephritis.

At the same time the question arises as to whether the nephritis might not be analogous to that form I have seen mentioned in Ashby and Wright's text-book on diseases of children, in which the nephritis is part of a general septic invasion, one might/

might say a pyaemia. It is described as occurring early in the course of anginous case, and is associated with marked albuminuria, a very slight degree of haematuria, but never dropsy nor uraemic phenomena. The author considers that it prepares the way for s subsequent attack of "past-scarletinal" nephritis. I myself am inclined to believe that, although toxins may have been responsible for some of the damage, there was an actual general invasion by some septic organism.

In two of my cases, IV.and VI, the condition was complicated by pneumonia. In the former it appeared immediately after the throat and mouth symptoms began to abate. Clinically it followed the course of an ordinary lobar pneumonia, with a definite crisis on the seventh day.

In Case VI., the pneumonia ran a more protracted course. It became evident a day or two after the throat condition, at the left base, and from this it spread upwards until, in two days, the entire left lung was consolidated. Nine days later, well marked dulness and tubular breathing were noted at the right scapular region. The temperature dropped by crisis on the eleventh day after the lung condition began. The physical signs rapidly cleared up except at the left apex.

The/

The association of lobar pneumonia with acute nephritis is a rather rare event in the convalescent stage of scarlet fever, yet I think it occurs frequently enough, and at so definite a period, that one is justified in refusing to consider it a mere accident. I have the notes of three such cases, all of which proved fatal.

C.W. aged 4 years was admitted suffering from a definite attack of mild scarlet fever, the temperature never reaching higher that 101°. The progress of the case was favourable until the 22nd day of the disease, when blood and albumen appeared in the urine, which was excreted in a diminished quantity. There was moderate general anasarca. On the following day marked dulness, tubular breathing and increase of vocal resonance was noted at the left base. Later the dulness became absolute, and the physical signs rather suggested the presence of fluid in the pleura; exploratory puncture, however, revealed nothing. Death occurred fourteen days after the initial rise of temperature.

At the post mortem examination I found excess of fluid in all the serous sacs, and in the tissues generally. The visceral pleura on the right side had a thick coating of recent lymph, and the lower lobe/

lobe of the lung on that side was consolidated. The consolidated lung tissue was of firm consistence, resisting considerable pressure, and was of a bright red colour. The kidneys were enlarged; the cortex was thickened, and the whole renal substance hyperaemic. The capsule stripped easily.

C.P. aged 3½ years was admitted with mild attack of scarlet fever. On the 22nd day of disease the temperature rose to 104°F., and blood and albumen appeared in the urine. On the next day a well marked pneumonia was found at the right base. The middle lobe of the right lung, and the base of the left were affected later. Subcutaneous haemorrhages appeared on the scalp, the right temporal region, and slightly on the legs. Death occurred eight days from the onset of pneumonia.

At the autopsy, the right pleural cavity was found to contain about three ounces of turbid serum; there was a thick layer of firm, recent lymph covering the visceral pleura over the lower and middle lobes. On section, the entire right lung except the anterior margin was consolidated, firm in consistence, and of a grey colour; a turbid fluid exuded on pressure. The left pleural cavity was normal, there was consolidation of the lower lobe on that side. The consolidation/

consolidation appeared to be spreading into the upper lobe from the root of the lung.

Cultivations from the lung on blood serum media gave a pure growth of streptococci.

The kidneys shewed no very definite morbid appearances, but there certainly seemed to be some general hyperaemia, with thickening of the cortical layer.

L.P., aged 6 years, after a moderate attack of the disease, developed nephritis on the 18th day. There was a quantity of blood and albumen in the urine, and the amount excreted was diminished. The temperature chart shewed the spiked appearance so frequent in acute scarletinal nephritis. On the 23rd day of disease, the respiration rate increased, and the remissions of temperature became less marked. Signs of pneumonia at the left base were evident, the whole lung eventually becoming affected. The child died a fortnight from the onset of the pneumonia.

The autopsy shewed extensive, thick, fibrinous deposit on the left pleura, some excess of fluid was present. The left lung was completelyconsolidated, of a grey colour, friable, and exuded muco-pus on pressure.

The kidneys were somewhat enlarged, with a thickened and hyperaemic cortex.

Cultivations/

Cultivations were only taken from the case C.P., the last one I saw. The other two cases occurred before I thought of the possibility of a close connection between pneumonia and nephritis, before indeed I was aware that they sometimes are co-existent.

An explanation for the different phenomena observed in these cases is, I think, most readily obtained by accepting the theory of a secondary infection, the true nature and cause of which, we at present are unable precisely to define. Von Jurgenson [11] discussing the "rare" case recorded by him, and which I have quoted, suggests a streptococcal infection as the most likely explanation. He expresses himself as follows, "In my opinion the simplest explanation is afforded by the presupposition of a sepsis, asserting itself very early in the joint pains on the fourth day of the disease. Besides the skin affection, there was probably a local involvement of the heart; also the pharyngeal complication can be interpreted as a streptococcus diphtheritis." "The condition is to be characterised as no more than a flaring up of the septic process." My cases exhibited all the evidences of a general sepsis that Von Jurgenson's did, except the erysipelas; at the same time two of them developed pneumonia, and another pleurisy/

pleurisy with effusion, all of which can be readily ascribed to the same cause.

I think we do not require to wait for the occurrence of these rare condition for examples of secondary infection in scarlet fever. Is it not likely that some of the commoner complications, such as nephritis and secondary adenitis, are due to the same cause, and that they all form local manifestations of a general septic invasion?

The onset of nephritis reminds one of the onset of many acute infections. We may get some warning of its approach, but frequently the temperature shoots up, the urine, which the previous day was clear on boiling is found to be loaded with albumen and blood.

The kidney condition, however, and the symptoms referable to it, although impressing the attention most, form only part of the clinical picture.

The glands of the neck in a very large percentage of cases are enlarged at the same time. This secondary enlargement does not appear to be thoroughly recognised in the general text-books, but no one can work long in a fever hospital without being struck with its frequency, and its close association with acute nephritis. Adenitis, unaccompanied by nephritis, does appear frequently at this time, yet in quite a large proportion of cases, blood and albumen/

albumen are found in the urine at the same time, or a day or two later. The inflammation often seems to be of a subacute type; the swelling may be very marked although pain may not be a prominent feature. I have found suppuration not to be so frequent as in the form that occurs at the early stage.

Caiger (12) distinguishes very definitely between the two types of adenitis; the early type associated with the acute throat symptoms, and analogous with that which occurs in any attack of sore throat, and the secondary, or as Caiger calls it "the primary pyrexial type," occurring at a later stage of the disease, and unconnected, as far as can be seen, with any local exciting cause.

Of 50 consecutive cases of nephritis, I found that adenitis is noted as present in 23; it preceded the albuminuria and haematuria in most of the cases. In this point my experience agrees entirely with the condition as Caiger describes it.

But besides this recrudescence of the inflammation in the cervical glands, which may have been infected at the early stage of the disease, widespread changes are described in the lymphatic system generally, at this time, and especially in association with nephritis/

nephritis. Von Jurgenson [13] first points out the early involvement of the lymphatic tissues, including the tonsils, spleen, and lymphoid elements in the intestine, and then goes on to say "in a secondary infection also, the spleen may be severely involved. Besides a simple hyperplasia, infarets and purulent foci of greater or less size may occur, and this forms no unusual occurrence." He also calls attention to an observation of Leichenstern, that an acute enlargement of the spleen associated with adenitis and nephritis is an event of no great rarity.

Caiger in Allbutt's System does not discuss the subject so fully, but he does say, that swelling of the lymphoid tissue in the intestinal mucosa is common, and is best marked in the solitary glands. He does not mention that this lymphatic hyperplasia may be associated with a nephritis.

Dr. Klein [14] has described very definite minute changes in the spleen, liver, and lymphatic glands, occurring in twenty-three cases of scarletinal nephritis, fatal in from the second to the eleventh week.

I have never been able to satisfy myself that there is any marked general adenitis in the early stage of the disease. In a few cases I have found some/

some slight enlargement of the inguinal glands, but it was not so marked that I could speak very positively on the subject.

With regard to the spleen, I have found a primary enlargement in a certain number of cases. Among 50 cases in which I paid particular attention to this point, I found the spleen to be palpable in 11, and in 13 cases the area of splenic dulness was definitely increased. The cases in which this enlargement was most definitely present, were of the severe anginous type.

I am afraid, however, that fallacy is very apt to creep into any clinical investigation on this point; a difference of opinion is common even among the most competent observers, unless indeed the spleen is definitely palpable, or the area of splenic dulness much enlarged. I think, there is no doubt, that, in post mortem examinations on patients dying from the anginous form of scarlet fever, some enlargement of the spleen is the rule, while in a large number of cases I have found well-marked enlargement of the solitary glands in the intestine.

I should like here to call attention to the fact, that, in the case of R.S., the first of my series, and the only fatal one in which an autopsy was made, circular punched out ulcers were found in the/

the mucous membrane at the lower part of the small intestine. Caiger (15) states that the enlarged solitary glands may ulcerate as in typhoid fever, but he does not say whether this occurrence is associated with the primary attack or a secondary recrudescence.

I shall give a brief acccunt of one remarkable case that I think may have some bearing on the subject. A girl, aged 4 years, was admitted to hospital on the 5th day of disease, suffering from Scarlet Fever of moderate severity. On the 8th day otitis media was noted on the right side. The temperature continued without any satisfactory explanation, the general condition of the child however was good, and the result of careful examination was negative. On the 17th day of disease abdominal pain was complained of, and on palpating the abdomen some widal enlargement of the spleen was noted. There was no widal reaction present. The discharge from the ear was very offensive; no mastoid tenderness and no symptoms pointing to intracranial complications were observed. There was a recurring general urticaria. The patient became gradually weaker, the temperature fluctuating between 99° and 103°. The dulness in the splenic region increased/

increased in extent until it extended at least three inches below the costal margin. There was occasional vomiting.

The intracranial complications of middle ear disease, abdominal tubercle, and typhoid fever were considered as possible diagnosis, but no definite conclusion was arrived at. The patient died on the 32nd day after the onset of scarlet fever.

Notes of the p.m. examination run as follows:-

Body:- Badly nourished; no external morbid appearances.

Lungs:- Pleural cavities are normal, nothing noticeable about the lungs except some congestion at the bases.

Heart:- The pericardium, endocardium, and cardiac muscle appear normal.

Abdomen:- On opening the peritoneal cavity, and lifting the great omentum, a large fluctuating swelling is uncovered in the left hypochondrium, it extends almost to the middle line, and comes considerably below the costal margin. On attempting to strip it from the ribs a large quantity (about a pint) of brownish yellow fluid escapes, of the consistence and appearance of moderately thick pus. The sac, when dissected out, is found to/

to be continuous with the capsule of the spleen. Only a third of the splenic tissue remains at the upper and posterior aspect, and in its substance there are several circular patches of a grey colour about the size of a large pea, they are firmer than the surrounding splenic pulp. The part of the spleen tissue in contact with the debris is soft, friable, of a brown colour, and evidently breaking down.

The other abdominal organs are normal.

The brain, meninges, and the sinuses are normal.

I am unable to suggest any diagnosis that satisfies myself. It was only when I read of the changes in the spleen in the later stages of scarlet fever, that I began to think, that, perhaps it was a case such as Leichenstern refers to, when he speaks of purulent foci in the spleen being one of the manifestations of secondary infection in that disease. In this case, however, there was no period of quiescence, and, with the exception of a trace of albumen in the urine, no other signs of a general septic intoxication were present. I think the question of pyaemia, the infecting focus being the middle ear, may be excluded. No sign of pus was found elsewhere, and the inflammatory trouble appears to/

to have been confined to the middle ear itself. But, is it not possible that the secondary infection, if it really was a secondary infection, came from that source?

If these complications are really due to a "septic" process to which the term secondary infection can be applied, what is the organism that is responsible? I think the bulk of the evidence is in favour of a streptococcus. Practically all authors consider that streptococci play a very important part in the patholcgy of scarlet fever, but there seems to be a great difference of opinion as to whether they are the only factor, or are superadded to some specific organism that has yet to be isolated. In my experience the predominant organisms in growths cultivated from the early scarlet fever throat are staphylococci, at the same time, we frequently find them accompanied by streptococci. In all the six cases I have described streptococci were very definitely present, almost to the exclusion of the other organism.

Jackmann [16] found that, out of 161 cases of scarlet fever, streptococci were present in the blood twenty-five times during life, pneumococci twice, and typhoid bacilli once. Streptococci were never isolated from the blood during the first two days of the/

the disease, nor from the blood of any of the fulminating cases. Their presence was always coincident with some secondary infection usually a tonsillitis. All the cases from which positive cultures were obtained ended fatally, death occurring within one or two days. The bacteriological examination of 70 autopsy cases shewed that, in a very large proportion, streptococcci were present not only in the heart's blood, but in the spleen, bone marrow, and less frequently in the kidneys. This infection appeared to be a secondary event in the course of the disease. The usual portal of entry was the tonsils. In all but one of the cases shewing streptococci in the blood during life, microscopic examination of the tonsils after death revealed very definite lesions, with accumulations of streptococcci in the blood vessels and lymphatics of surrounding tissues. The exception was a case of otitis media with mastoid infection. The author concludes that streptocccci play a very important role in scarlet fever, although the condition may have no etiological significance and must be considered a secondary event.

I have ventured to refer at some length to Jackmann's article, as it agrees so well with the inference I wish to draw from the cases under discussion/

discussion.

Pearce [17] also refers to the frequency with which streptococci are found in the "secondary" inflammatory lesions, but, unlike Jackmann, he is of opinion that in the primary general infection they also play a very important part.

I think we must certainly consider the tonsils to be the focus from which these secondary infections most frequently start. The regularity with which we see cervical adenitis ushering in an attack of acute nephritis points strongly to this conclusion. It is true, as a rule, that we do not see inflammatory changes in the tonsils themselves, but the obvious path for infection is from the tonsils, unless we are prepared to believe that the infecting virus has lain dormant since the primary attack subsided.

Those cases in which there is a return of the early faucial inflammation, and which are usually described as cases of secondary tonsillitis, are not necessarily followed by nephritis. A reasonable explanation of this would be that the lymphatic sieve was sufficient to save the blood from general infection. I should be inclined to consider the six cases I have described as being an infection with a/

a very virulent type of organism, occurring perhaps in a subject of low resisting power.

In this particular, however, it is remarkable that secondary tonsillitis should be more frequent in adults, and should occur just as often after mild attacks of the disease, in which no particularly heavy demand had been put upon the patient's recuperative powers.

My experience in this point coincides with that of Dr. Caiger and Dr. Bowie, who both remark that tonsillitis is more frequent in older patients, and is not necessarily preceded by a severe primary attack.

The buccal cavity, especially the teeth and gums, must be considered as another possible source of infection quite apart from the tonsils. Dr. William Hunter, in a paper I have previously referred to, lays stress on the view that "oral sepsis" not only causes a more severe primary attack of the disease, but also renders the patient more liable to complications. By oral sepsis he evidently means decayed teeth, and septic foci that are apt to develop in the contiguous soft tissues. The complications that he finds most frequent in this class of case are "secondary" angina, nephritis, and rheumatism/

rheumatism. He considers it to be a streptoccccal or staphylococcal infection superadded to the specific virus of scarlet fever.

The publication of this suggestion caused some indignation among the Metropolitan Asylum Board authorities, as Dr. Hunter contrasted the death rate of .8% in the London Fever Hospital with theirs which amounted to 3%. In a subsequent letter, (18) however, he pointed out that the patients of the London Fever Hospital are drawn from the better classes, which are accustomed to take better care of their mouths. He also explains that he does not refer to the stomatitis that so often complicates severe scarlet fever, but to the oral sepsis present before the attack.

For my own part, I am inclined to doubt the extreme importance of this pre-existing oral sepsis, That its existence does determine, to some extent, the severity of the primary attack and the frequency of the complications I am prepared to admit, but I cannot see how we can thus explain the difference between a death rate of .8% and 3%. I think that the general health of the individual, his surroundings at home, and especially his treatment during the two, three or four days before admission, would provide a more/

a more adequate explanation. In a hospital of this size, in a large industrial city, we see many patients whose teeth and mouth are in a very septic state, yet in none of the six cases I have recorded was dental caries a marked feature.

Besides the ulcerative stomatitis, so common in the early stage of severe scarlet fever, I have noticed another buccal condition that may be severe enough to call a stomatitis. I have the notes of five cases in which it was very well marked, but in its milder forms I have seen it more frequently. The most definite examples have occurred in adults and older children. Its onset coincides with the onset of an attack of acute nephritis; it may precede it and probably does, as my attention was usually drawn especially to the case by the nephritis. The breath is "heavy," one could not say it is offensive. There is marked injection and some swelling of the gums, with a tendency to bleed; this however does not occur spontaneously, but after any manipulation about the mouth. Sordes are apt to collect on the teeth. The patient does not suffer much inconvenience, although there may be some tenderness on tapping the teeth. It does not appear to be associated with dental caries. In two of my cases there was cervical adenitis of the chronic/

chronic type, in which the swelling was marked but pain was not severe; the condition cleared up slowly without suppuration. The mouth improved very slowly on treatment with astringent and antiseptic lotions; I could not definitely say, however, that the improvement in the stomatitis was immediately followed by an improvement in nephritis, indeed the urine in some of these cases was particularly long in clearing up. It is, I think, quite reasonable to suppose that the buccal mucous membrane in these cases was the seat of infection.

Gallois (19) has advanced the opinion that it is in the naso-pharynx we must look for a septic focus. He thinks that to avoid renal complications the upper respiratory passages must be carefully disinfected, and, as soon as the naso-pharynx has recovered, the strict milk diet can be departed from, without the risk of setting up nephritis.

I only read this a short time ago, and since then I have been looking out for cases that seem to be examples of this source of infection. A case has just come under my notice that seems to lend some support to the view. A girl, aged 4 years, was admitted to Hospital suffering from an attack of scarlet fever of moderate severity. The recovery from/

from the early symptoms was complete and the patient was allowed up. On the 22nd day of disease the temperature rose to 101.6°F, and a profuse muco-purulent nasal discharge was noted. The discharge caused excoriation of the nostrils and upper lip. Cultivation shewed a growth of staphylococci; streptococci were not present. On the succeeding day the condition of affairs was unchanged except that a moderately heavy trace of albumen was present in the urine. On the third day the urine contained a cloud of albumen on boiling, and enough blood to give it a decidedly smoky appearance. At the time of writing - a fortnight later - the nasal discharge is better, but the urine still has a trace of albumen and a faint trace of blood. I think the question is one quite worth paying attention to.

I have attempted to establish some relationship between otitis media and nephritis from the hospital statistics, so many of the cases with otitis however have at the same time some of the other evidences of sepsis that I have alluded to, that it is very difficult to draw a positive deduction. I have seen individual cases that seem to point very strongly to the conclusion that the middle ear is sometimes the infecting focus. I have one case in particular in my/

my mind. The ear discharge commenced early in a moderately severe primary attack, from which a good recovery was made; nephritis set in early in the fourth week, and was accompanied by a painful enlargement of the gland which lies in front of the mastoid process and below the ear. The blood and albumen disappeared from the urine at three different times, their reappearance on the first two occasions was preceded by an acute exacerbation of the middle ear trouble, with recurrence of the adenitis, at no time was there any swelling or tenderness over the mastoid process itself.

I have examined the notes of 112 cases of nephritis and severe secondary albuminuria, occurring in 1905, to see in what proportion of cases could a definite septic focus be found. I accepted those cases in which there was otorrhoea, persistent rhinitis, secondary adenitis, and secondary tonsillitis. In 46 cases (41%) none of these complications accompanied the nephritis, while in 66 cases (59%) one or more was very definitely present.

It must be remembered that the notes were taken in the ordinary routine of ward work, and that the patients were not examined expressly with the idea of finding some possible infecting source, in many cases, perhaps/

perhaps, not itself of much moment. If a large number of cases were examined with this end in view, I think it quite likely that a much larger portion of nephritis cases would be found to have some inflammatory trouble in the nose, naso-pharynx, fauces or middle ear. At the same time we must not forget that a severe primary attack is more apt to leave behind it some of these conditions, and that possibly it was the fact of it having been a severe attack that rendered the patient more liable to the secondary infection.

I have not referred to some of the other complications of scarlet fever, as I have been unable, in the cases coming under my notice, to find any connection between them and the other secondary phenomena occurring about the end of the third week of the disease.

"Scarletinal rheumatism," in its commonest form, is in my experience one of the early complications, occurring while the initial inflammation of the fauces lasts, and before the rash has faded. Caiger (20) has found it to be most frequent about the fifth and sixth day. It certainly does occur later, but I have never noticed its association with nephritis, apart from those cases I have recorded where, besides the kidney condition, there was present also/

also an intense inflammatory and gangrenous condition of the tonsils, besides other evidence of a general toxic absorption. Caiger,(21) speaking of the association of nephritis with joint pains, says that "the nephritis is often associated with adenitis and not infrequently with rheumatism which usually precedes it." If he means that the "rheumatism" precedes the nephritis by ten days or thereabout, I would not be prepared to say that my experience differed from his. There was no close association of these two complications among the 112 cases of nephritis I have mentioned.

Endocarditis, which I have found to be a rare complication, appears also to be more likely to occur in the early stages. I cannot say I have ever seen an undoubted case associated with nephritis. The difficulty of making up one's mind as to the presence of endocarditis in scarlet fever is great; mitral murmurs are not uncommon,but they appear to be for the most part "functional" or at least haemic. I have made or been present at over 100 post mortem examinations of cases of scarlet fever, and endocarditis was not present in any of them. Two of the six cases I have recorded at the beginning had well marked mitral systolic murmurs. It was best marked/

marked in the fatal case H.B. (No.V.) in which I unfortunately was not able to get permission for an examination. I examined the other case M.W. (No.VI.) six months after discharge from hospital, and found no signs of cardiac disease.

Chorea, another admittedly rare complication, - I have seen four cases asscciated with scarlet fever- appears to occur earlier than the critical period at which nephritis, adenitis, etc., do; I have never seen it asscciated with them.

I have sttempted to make out a case in favour of classifying the six cases I first mentioned, along with the other complication of scarlet fever occurring particularly at the end of the third week. The evidence I have been able to bring forward is, I am afraid, only circumstantial, still I think it gives me some ground for coming to the conclusion that, at this period, patients convalescent from scarlet fever are liable to a septic, most likely a streptococcal infection; and that the infecting focus is most frequently in the tonsils, which may themselves undergo inflammatory changes, but cervical adenitis may be the only evidence of it; other sources of infection may be the mouth, the nose, and the middle ear.

In/

In my cases, I would suggest that the local manifestations at the infecting lesion were particularly severe, either due to the virulence of the organism or the susceptibility of the patient.

I have never read nor heard of any suggestions to explain why convalescents should be so liable to these complications at this particular period.

I felt rather diffident about describing these cases in a thesis, seeing that none of the regular text-books refer to any condition resembling them; after reading, however, the cases described by Von Jurgenson and Symes, I considered myself quite justified in recording them, and attempting in some way to explain the phenomena observed.

I have said very little of the treatment that was adopted, simply because our efforts were confined to attempts to control the gangrenous process in the mouth and throat, and to sustain the patient's strength by suitable feeding.

Probably any of the other unirritating antiseptics would have done just as well as the proprietary coal-tar preparation Izal, but I have found it a very reliable agent for treating conditions of this sort. Whether the treatment influenced the course and extent of the lesions I cannot/

cannot say. The inflammatory process seemed in all the cases to run a definite course, like so many acute infections, so that after three or four days, when the patient seemed in extremis, a rapid improvement would set in. In this respect the condition was quite different from the gangrenous stomatitis occurring after debilitating diseases, of which I have seen two cases in children convalescent from typhoid fever, where no treatment either of an active or more conservative character seems to influence the progress of the disease.

In all the cases the faucial condition, along with the pneumonia in two of them, most arrested the attention, and treatment was directed accordingly. The nephritis, as is often the case after scarlet fever, did not give rise to very marked symptoms, and the usual remedial measures were not adopted until the more urgent features had disappeared.

I have no experience of the use of anti-streptococcic serum in scarlet fever. The reports published in the literature are certainly not very encouraging, at least when the ordinary serum is used. Of recent years, however, special sera have been prepared by different observers, the animals used in their preparation being injected with organisms/

organisms obtained from fatal cases of scarlet fever. Moser [22] reports very good results from a serum he prepared by injecting a horse with living cultures from different cases; the primary throat lesion appeared to be lessened in severity, and the whole course of the disease was shortened. He says nothing about its effect on the renal and other secondary complications. Baginsky [23] thinks that the serum of Aronson gives better results. It differs from Moser's serum, in that the virulence of the streptococcus is exalted by first passing it through susceptible animals before inoculating the horse. If the secondary complications are really due to a streptococcal infection, a serum of this special character might give good results.

I now wish to consider the subject of scarlet fever in relation to haemorrhagic and purpuric phenomena, in the first place, in the early stage of the disease.

Great diversity of opinion appears to exist as to the frequency of haemorrhages into the skin and from the mucous membranes in the early stage. Osler [24] classifies scarlet fever into Simple and Malignant, and under the head Malignant he places three forms/

three forms, (1) Atactic, (2) Haemorrhagic, (3) Angiose.

He says that in the Haemorrhagic form the rash is haemorrhagic almost from the first, there is bleeding from mucous membranes, and death takes place from profound toxaemia in two or three days. He has seen two cases of this form in robust adults, but more commonly it occurs in debilitated children. Many of the text-book accounts do not mention this form of the disease; Caiger's classification, for instance, is Simple, Malignant and Anginous.

Dr. Gee [25] speaks very definitely on the subject; he says, "Scarletina Haemorrhagica is so rare, that the occurrence of a passive haemorrhage from several mucous membranes at once, might make one with justice suspend the diagnosis, until the notion of possibly having to do with variolous roseolae was discarded."

I have never seen a case that conforms to Osler's description; several have come under my notice that could be classified under the heading "Malignant," but in none of these could the rash be called haemorrhagic. I have found small petechiae to be not uncommon in acute attacks, in which the rash is very vivid; they are most evident, as a rule, under the/

the clavicles and at the flexures of the joints, and may remain visible for some days after the hyperaemic element in the rash has disappeared. I have also noticed that in this class of case a slight trauma may produce a certain amount of extravasation into the superficial layers of the skin. These cases, although at the onset of an acute type, are not necessarily followed by ulceration and the other features of anginous scarletina, the temperature may drop rapidly in a day or two, and the convalescence may be uneventful.

I have only seen two cases in which haemorrhagic symptoms were, in the early stages, a marked feature. Both cases on admission presented the appearance of Anginous Scarletina in a severe form; the haemorrhages appeared shortly afterwards. The fact that, on cultivating the throat of one of the cases, a bacillus indistinguishable from the bacillus diphtheriae of Loeffler was found, adds further interest. The notes of the cases are as follows:-

I. P.V. Male aged 3½ years. Admitted to Hospital on the 4th day of disease, 19th June 1906.
June 19th. Fauces are markedly injected and swollen, and there is some soft easily detached exudation on the tonsils. The tongue is heavily furred. Moderate cervical/

cervical adenitis on both sides. General well-marked punctiform rash.

June 20th. Patient very restless at night, takes nourishment well. Examination of heart and lungs reveals nothing abnormal.

June 21st. Again a restless night. Adenitis very marked on both sides.

June 22nd. Profuse muco-purulent nasal discharge. Quantity of soft, yellow exudate on the tonsils. Cultivation, a growth of cocci.

June 24th. Deposit has cleared off the tonsils, leaving extensive ulceration spreading to the soft palate and uvula. Taking food better and sleeping better.

June 27th. There is a perforation of the soft palate on the right side. Restless again last night.

June 29th. 10 a.m. Quantity of haemorrhage from the mouth this morning, there is no definite bleeding point. Swelling and ulceration of the faucial structures are very marked.

2 p.m. A purpuric rash has appeared since morning visit. The haemorrhages are cutaneous, varying in size from a pin-head to a pea. They are most numerous on the front of the thorax and abdomen, but are present also on the flanks and back.

On/

On the front of the chest they tend to coalesce and form larger areas. Several subcutaneous ecchymoses, about the size of crown pieces, are present in the sacral and lumbar region; on each temple also, there are haemorrhages of the same character. Blood is oozing continuously from the mouth. The nasal discharge is sanguineous. There is no enlargement of the spleen. Examination of the heart, lungs and abdomen reveals nothing. Blood count. Red cells, 3,600,000. Leucocytes,18,700.

June 30th Haematuria and Albuminuria. Subcutaneous haemorrhages on the back increasing in extent.

July 1st. Condition of the patient certainly worse. Pulse is feeble and soft. Respirations hurried, moist sounds at both bases, no consolidation. Haemorrhagic discharge from the mouth and nose continues. Haemorrhages on the front of the trunk unchanged, they are rather more numerous on tha back.

July 2nd. Subcutaneous haemorrhage on the right cheek. There is very little bleeding now from the mouth and nose. Melaena.

Patient died on the evening of July 2nd.

I unfortunately was unable to obtain permission for an autopsy.

II./

II. E.P. Male, aged 6 years. Patient was admitted on the 3rd day of disease suffering from a very severe attack. There was nothing remarkable about the history of onset.

November 26th 1906. The fauces are injected and swollen, there is some soft exudate. Tongue, typical strawberry. Marked double cervical adenitis. General vivid punctate rash.

November 27th. Delirium. Petechiae all over the trunk, extremities, face and head. Marked ulceration of the tonsils. Crepitations at the bases of both lungs, no consolidation. Pulse very feeble and rapid. Heart, nothing noteworthy.

11 p.m. Patient very restless, complainging of sore throat. Profuse muco-purulent nasal discharge.

December 1st. Resting better. Tongue and fauces cleaner.

December 4th. General condition of the patient unchanged. Slight epistaxis after syringing the nose this morning.

December 5th. Haemorrhagic discharge from the mouth and nose. Large subconjunctional haemorrhage in left eye, smaller one in the right eye. Cutaneous ecchymosee above the bend of the left elbow, and on inner side of both thighs. Numerous small haemorrhages/

haemorrhages, varying from the size of a pin-head to a small pea, scattered over the trunk. Restless night.

Cultivation shewed pure culture of bacilli having all the characters of diphtheria bacilli.

December 6th. Small amount of black melaena. Ecchymoses on the back are more marked. Blood is still oozing from the mouth and nose. Gingivitis. Cultivation taken again with same result.

December 7th. Vomited "coffee grounds" in the night. Right and Left otitis media.

December 8th. Slept fairly well. Subcutaneous haemorrhage in front of the left ear. Numerous cutaneous haemorrhages still appearing over the trunk and on both legs. Gums fungating, lower incisors are loose, blood oozing continuously from the mouth. Breath is offensive, but not particularly so.

December 10th. Conjunctival haemorrhage is not so marked. Melaena still present. Discharge from the left ear is haemorrhagic. Much less blood from the mouth and nose. Condition of the gums perhaps better. Complaining of pain in the region of the right shoulder, there is marked swelling and tenderness on the outer side of the joint, no fluctuation. The joint moves quite freely, and is evidently/

evidently itself unaffected.

December 13th. Swelling over the deltoid is tense. Thin offensive pus on incision. There is a large cavity running up behind the joint, the joint itself appears to be free.

The patient improved very much after this; the temperature, which had been running between 101°F and 104°F, fell until it reached normal on December 18th. All haemorrhages disappeared, and, with the exception of some subacute cervical adenitis, nothing was noted. On the evening of January 9th the temperature rose to 103.6°F, and acute pain was complained of in the left ankle. On incision the following day, pus was found in the joint. A few days later both elbow joints became distended and painful, they likewise were incised and pus evacuated. Later still the left knee joint became involved. The patient eventually died from exhaustion on January 27th. Permission for an autopsy could not be obtained.

In both cases the usual treatment by swabbing and irrigating the throat and nares with mild antiseptics was carried out, and in addition, ten grain doses of chloride of calcium were given every four hours. In the second case ten **minims** of Burrows/

Burrows and Wellcome's solution of adrenalin chloride were administered. I am afraid that, on such slender evidence, one can hardly give all the credit of the temporary improvement in the second case to the calcium chloride. Still the boy's condition seemed to improve immediately on its exhibition. It was not the first time I tried to persuade myself I had got some tangible result from the use of this drug. As for the adrenalin, I am very sceptical about its usefulness when given internally, especially in cases in which we must presume that it is the coagulating power of the blood that is at fault.

Under which of the forms of scarlet fever are we to classify these cases? They certainly ran quite a different course from the haemorrhagic scarlet fever that Osler and some English writers have described, and that many have found so rare.

The onset and the first two or three days presented no features that differed in any way from the very common type of the disease, the anginous form. It was only when the gangrenous and septic condition of the throat became extreme that the altered state of the blood, caused by the toxic absorption, made itself felt. Von Jurgenson, in Nothnagel's/

Nothnagel's text-book, says, in this connection, that "with the exception of cases caused by sepsis and gangrenous intoxication, haemorrhagic scarlet fever in the narrow meaning of the term is quite rare." Jenner's opinion quite coincides with this view, that by far the commonest form of haemorrhagic scarlet fever is found in cases where, after several days of "Angina and severe fever," the rash becomes petechial, then purpuric, and after death, which occurs in a dew days, haemorrhages are found into the serous and mucous membranes, the spleen is enlarged, the blood is very thin, and the organs otherwise normal.

Why we do not see this toxic purpura oftener in Scarletina Anginosa I cannot suggest, nor is it easy to say what was the determining factor in these cases.

In the second case a very interesting question arises. Were the symptoms due wholly or in part to associated diphtheria? Von Jurgenson throws some doubts on the genuineness of some cases of purpura, occurring in the later stages, described by Henoch, as no bacteriological report was, at that time (1878) possible. Dr. Bliss [26] also, referring to a case of purpura haemorrhagica at a later/

later stage of the disease, gives it as his opinion, that it is doubtful whether extensive subcutaneous haemorrhages ever occur at the early stage of scarlet fever, and that those that have been described were possibly due to haemorrhagic diphtheria.

I personally think that the haemorrhages were not due to the toxaemia of diphtheria. My reasons for holding this view are, I believe, quite plausible. The patient never at any time exhibited symptoms that in any way suggested diphtheria, the condition of the thorat was that which one sees so frequently in severe scarlet fever. The course of the disease was not that which haemorrhagic diphtheria , as I know it, follows; the temporary improvement, the absence of cardiac symptoms, and of paralysis, were all against a diagnosis of diphtheria intoxication. Quite early in the disease the abscess over the shoulder joint was noted, shewing the probable existence of the general sepsis which, in my opinion, was responsible for the purpura. It is by no means uncommon to have cases admitted to hospital that, on cultivation, shew the presence of Loefflers bacillus. These cases are of course always isolated, yet it very rarely happens that the subsequent clinical course of the disease confirms the diagnosis/

diagnosis that one might feel inclined to make from the bacteriological examination.

I think one is quite justified in considering these cases to be examples of toxic purpura, of a quite distinct character from that very rare condition described as Scarletina Haemorrhagica.

It is not possible that in one case, the tendency to haemorrhage is occasioned by the specific virus of scarlet fever, while in the other, the septic organisms that later come into play cause the damage?

Another form of purpura, occurring at about the 18th to the 25th day, has been described. I have met two cases, which at the time of their occurrence seemed very puzzling. They are not exactly like the published cases, but if one does not accept this explanation I am at a loss to account for them.

Post-scarletinal purpura is not mentioned in any of the English text-books. Caiger, Goodall and Washbourn, Ashly and Wright, Collie and others are silent on the subject. Henoch[27] appears to have been the first to recognise the fact that such a thing does occur. In his Lectures on Diseases of Children he mentions the "haemorrhagic diathesis" as a sequela of eight cases; in all, the symptoms were first/

first noticed in the 3rd or 4th week, and all terminated favourably. In four, nothing was observed beyond blood extravasation into the skin in different parts of the body, in others, there occurred at the same time haemorrhages from mucous membranes, particularly the nose. Some of the cases were complicated by nephritis.

I have found five cases described in the Ebglish literature on the subject.

Dr. Banks (28) reports one case from the Grove Fever Hospital, London. A youth, aged 17 years, convalescent from a moderate attack of scarlet fever, on the 20th day of disease complained of pains in the shoulders, wrists, lumber region, and in the epigastrum; vomiting was severe. Large haemorrhages appeared in the skin and subcutaneous tissues over the elbows, and hands. There were three separate attacks of haematemesis. The result was recovery. The treatment adopted was Calcium Chloride in doses of five grains, and morphia.

Dr. Bliss (29) reports another, where the patient, aged 3 years, was sent to the Grove Hospital as a case of diphtheria. He had had antitoxin injected but was an undoubted case of scarlet fever. The date of the onset of disease is not clearly stated./

stated. He was brought to Hospital on May 16th and the haemorrhagic symptoms appeared on June 14th, so I think one may presume that it was about the end of the third week or the beginning of the fourth. Extensive haemorrhage occurred into the skin of the trunk, limbs, eyelids, and from the stomach and rectum. Death occurred inside 36 hours. Dr. Bliss thinks that the antitoxin had nothing to do with the purpura.

The third case is recoded by Dr. Cullen.(30) The patient became ill on February 10th 1903, and safely passed through a moderate attack of scarlet fever. On the 20th day of disease there was pyrexia, and a rash developed that was diagnosed as that of rötheln. On the same evening headache, vomiting and albuminuria were noted. On the following day the patient complained of pains in the legs, and purpuric patches appeared on the legs, thighs, and palms of the hands. The whole of the subcutaneous tissues of the legs eventually became infiltrated with blood. Haemorrhages were also present on the arms, ears and cheeks. The patient died 72 hours after the onset. of symptoms. The treatment adopted was the administration by the mouth of Calcium Chloride, ergot, and suprarenal extract.

The/

The post-mortem examination revealed, besides the haemorrhage into the subcutaneous tissues, extravasation into the mediastinal tissues, under the pleurae, between the layers of the gastro-splenic omentum, and into the areolar tissue surrounding the kidneys.

Dr. Cullen remarks that during nine years, only two cases of purpura haemorrhagica, following scarlet fever, are recorded at the Fountain Hospital.

The fourth case came under Dr. Collie's [31] notice at the Eastern Fever Hospital, Homerton. It does not differ in any great degree from the others. The patient was nine years of age. The onset of the scarlet fever was on October 20th, and the purpura was noted on November 10th, about three weeks from the initial symptoms. There was marked extravasation into the subcutaneous tissues of the arms, legs, and ears. The patient complained of acute abdominal pain, this was accompanied by intractable vomiting.

The post-mortem examination revealed congestion of the tonsils, marked anaemia, but no haemorrhage into the internal organs, and no enlargement of the spleen.

The fifth case is recorded by Dr. Sidney Phillips [32] from the London Fever Hospital. It was associated/

associated with recrudescence of the throat symptoms, and with pain in the fingers, wrists, and ankles. There was haematuria, epistaxis, and petechiae on the limbs and chest. The patient eventually recovered.

Henoch mentions that cases have either been published or communicated to him by Strŏm, Arctander, Charron, and Michaelis.

One of my cases conforms fairly well to those I have quoted, especially to that described by Dr. Cullen. The patient, a female aged 16 years, first fell ill on September 12th with the usual history of headache, vomiting, sore throat and rash. On admission the diagnosis of scarlet fever was agreed with. The temperature after admission was never higher than 100.4°F, it reached normal two days after admission. On the morning of the 14th day of disease, the patient complained of pains in the hands. Redness, swelling and tenderness of the metacarpo-phalangeal joints was noted, and salicylates were ordered. On the evening of the same day she complained of general abdominal pain, and vomited some green fluid. The abdomen was examined with negative results. The further notes of the case run as follows:-

September 22nd. Has been complaining of abdominal pain all morning, it is referred mostly to the left hypochondrium/

hypochondrium. Vomiting quantities of turbid green fluid. Marked abdominal succussion. Abdomen moves freely on respiration. Liver dulness is absolute. There is no dulness in the flanks. The spleen is not enlarged. By percussion the outline of the stomach can be marked out, the greater curvature extends to the umbilicus. Slight pressure in the epigastrum causes splashing sounds to be communicated from the aorta. The urine on boiling shews a cloud of albumen. Patient complains of sore throat, there is some injection of the faucial structures generally.

September 23rd. Vomiting continues, but not so persistent. Still complaining of pain in the left lower costal region, also some general abdominal tenderness. Complains of sore throat; there is well marked injection of the tonsils. A heavy trace of albumen in the urine. Temperature 100°F.

September 24th, 11 a.m. Slept occasionally through the night. Still vomiting dark green fluid. Patient is evidently worse. Abdominal pain is still complained of.

6.30 p.m. Has not vomited since lavage was practiced this morning. Pulse feeble. Looking very ill. Complaining of acute abdominal pain, and also of sore throat; examination of abdomen reveals nothing, the/

the fauces are distinctly injected, there is slight exudation, Well marked extravasation of blood into the tissue of each ear. The patient became rapidly weaker and died at 9.30 p.m., four days after she first complained of joint pains. The treatment was directed principally to relieve the vomiting; sinapisms and fomentations were applied to the epigastrum, lavage was carried out, and bismuth was given by the mouth. In the later stages the patient was stimulated with brandy and strychnine hypodermically.

The following possible diagnoses were in turn suggested and discussed without a definite conclusion being arrived at:-

(1) Obstruction of the intestine, high up.

(2) Uraemia.

(3) On the appearance of the haemorrhages into the tissues of the ears, especially as they were associated with a throat condition, the possibility of an acute general intoxication was hinted at. The throat condition, although complained of, and quite definitely present, was not one of the marked features of the case.

The post-mortem examination was made twenty-four hours after death. The notes run as follows:-

Body/

Body is well nourished. Definite desquamation in the hands and neck. Several subcutaneous haemorrhages, about the size of a pea over each knee. (These were overlooked during life). Haemorrhage into the lobule of each ear.

Fauces:- Some yellow exudation in the crypts of the tonsils.

Heart and Pericardium:- Normal.

Lungs:- Congestion at the bases. The blood in the vessels is of a dark colour, not clotted, and syrupy in consistency.

Abdomen:- The stomach is distended, the greater curvature extending to 1½ inches below the umbilicus. The small intestine is pushed down into the pelvis. The stomach walls are thin. The contents are dirty grey in colour and liquid. There are a few petechiae in the mucous membrane, towards the pyloric end. The walls of the duodenum appear to be infiltrated with extravasated blood, this is most evident on viewing the gut from the peritoneal side. The haemorrhage is evidently into the muscular coat. There is haemorrhage into the areolar tissue in the region of the duodenum. In the lower part of the ileum the same condition of affairs is present, in an even more marked degree.

A/

A few of the Peyers patches shew haemorrhagic points. The other abdominal organs appear healthy.

The other case does not follow so closely the course of the described cases, still I think that, in the absence of any other explanation, we must accept purpura fulminans as the most feasible diagnosis.

The patient, a boy aged four yeats was admitted to Hospital on March 26th 1906, the 6th day of disease, suffering from a quite definite attack of scarlet fever of a mild character. The temperature never rose abcve normal. The heart and lungs were normal, and the urine contained ncthing abnormal. The patient was allowed up on the 14th day of disease, and was out in the airing-court on the 18th day. On the evening od April 15th, the 24th day of disease, slight cervical adenitis was noted. On the following day the adenitis was more marked especially on the left side. There was some swelling and injection of the tonsils. The boy passed a comfortable night, not complaining of anything. He was reported to have "vomited" about two ounces of blood in the morning; on examination some swelling of the fauces was still present, there was no ulceration. Some blood clot was clinging to the molar teeth. I was called/

called to see him at 12.45 a.m. on the night of April 17th, and found him vomiting large quantitied of blood. He died within half an hour afterwards.

Post-mortem examination twelve hours later:-

Body:- Well nourished. Nothing noticeable externally except a haemorrhagic discharge from the mouth and nose.

Fauces:- The tonsils are perhaps somewhat swollen, but there is no appearance of any ulceration from which profuse bleeding could have taken place. The cervical glands, especially on the left side, are enlarged and indurated, there is no suppuration.

Abdomen:- There is a large quantity of fluid blood in the stomach; no ulceration nor erosion found on its mucous surface. Blood is mingled with the contents of the small intestine and colon, but no source of haemorrhage found.

The Kidneys, lungs, heart and spleen are normal. Cranial cavity not examined.

I think there is no doubt that the first case I have described was one of post-scarletinal purpura fulminans, as Henoch calls it. I grant that the cutaneous haemorrhages, that were so marked a feature of the recorded cases, did not occur with anything like the same severity, yet they were definitely present; while the haemorrhage into the wall/

wall of the gut and the extraperitoneal cellular tissue was remarkable, and had its counterpart in the extravasation into the gastro-splenic omentum and perirenal tissue in Dr. Cullen's case.

The onset in my cases, like the others, was acute, after a period of apparent good health. The first symptoms in all, were noted in the third week of the disease. The occurrence of joint pains in the early stage was, I think, an interesting feature, they are recorded in two of the five cases I have quoted. Vomiting was noted in three of the cases, and in two the patient, as in my first one, complained of acute abdominal pain. Other points worth drawing attention to are, the recrudescence of the faucial inflammation in Dr. Cullen's and Dr. Phillip's cases, and the occurrence of haematuria or albuminuria in two of the cases as well as in mine. The condition in all instances was evidently a very serious one; two recoveries are mentioned, but only after a very alarming illness. It is remarkable that none of Henoch's eight cases proved fatal, as in the main points they appear to coincide fairly well with those I have mentioned. This discrepancy has been noticed by more than one of those who have recorded cases.

With regard to my second case, it certainly presented/

presented features quite apart from the others. No cutaneous haemorrhages occurred, but alarming haematemesis was the symptom that most arrested the attention, and haemorrhage from the mucous membranes was not severe in any of Henoch's cases, while in only one of the later cases was it to any extent noticeable. Two features, however, not in themselves of much moment, but important in giving some clue to the true nature of the case, were present, namely, the time of onset, and the occurrence of secondary adenitis.

Various explanations for this very rare complication of scarlet fever had been advanced. Henoch confesses his inability to account for the symptoms on a rational basis, but suggests that the scarletinal toxin may cause some "molecular change" in the walls of the small blood vessels, producing a greater liability to rupture. Dr. Sidney Phillips thinks that possibly the scarlet fever in the rheumatic subject may cause the occurrence of haemorrhages. In his case the joint pains were a very marked feature, and there was a very strong family history of rheumatism. Another possible explanation, and one that perhaps better meets the facts, is to consider the purpura as the manifestation of some blood change, the true nature of which we are/

are at present ignorant, brought about by some acute intoxication. If this be true the purpura comes under the same category as acute nephritis, secondary adenitis etc.

I think the evidence in favour of this view is very strong. A recrudescence of the throat and glandular inflammation was present in both my cases, as well as in two of the others; if the faucial condition were not very marked one can easily understand it being overlooked, as the purpuric symptoms were usuallyso alarming. If the patient were young this would be still more likely to occur.

I might almost have classified these two cases along with those in which there was gangrenous tonsillitis and stomatitis. The difference between them was possibly only a matter of acuteness of intoxication. In one instance the condition of the buccal cavity and the pharynx - the infecting scurce dominated the whole case, but nevertheless sufficient intoxication took place to cause the haemorrhagic rash on the extensor surfaces, and to set up an acute nephritis; while in the other, the virulence of the infecting organism was so great, or the resisting power of the individual so low, that the local manifestations had not time to become marked, and the/

and the patient succumbed to an acute sapraemia or septicaemia, of which the principle feature was malignant purpura.

With regard to the treatment, I am afraid we have no drug from which one can expect a very definite result. None of the reputed anti-purpuric remedies or styptics were tried in my cases. In the first one, the haemorrhage into the tissues of the pinna was only noticed shortly before death, when the patient was evidently in extremis, and only then was the true nature of the case suspected; while in the second one, with the exception of the cervical adenitis and slight "haematemis," there were no symptoms to suggest that the patient was seriously ill, much less than death was to occur so shortly afterwards from profuse haemorrhage. Calcium chloride, adrenalin, or some of the other internal remedies might have been tried, but probably all would have been equally useless.

The occurrence of "Relapse" in scarlet fever has in recent years attracted a good deal of comment. In most of the text-books on general medicine its occurrence is still ignored, but in the larger books, and especially those that pay special attention to infectious/

infectious diseases, a considerable space is devoted to the subject. In the journalsfrequent communications are found from practitioners who have met the condition, and have considered the event worth publishing. Anyone who has worked for a time in the wards of a large fever hospital must have met some cases.

As I have said, the condition appears to have received due recognition only in recent years. Collie (33) considers it to be exceedingly rare. Henoch (34) says that relapses certainly do occur, although not so frequently as in typhoid fever. Caiger has met it in .7 per cent of 12,000 cases. The M.A.B.Hospital reports give its incidence as 1.73 per cent among 10,520 cases in 1904, and 1.94 per cent in 16,326 cases in 1905.

Our experience at Lodge Moor Hospital does not differ materially from the figures given by Dr. Caiger. During the years 1905 and 1906, 5323 cases were treated, and among these there occurred 14 cases of relapse, giving a percentage of .26. All cases were rejected in which there was any doubt about the genuineness of the first attack.

Abbreviated/

Abbreviated notes of the cases are as follows:-

1. T.N. Male. aet, 8 years. First attack mild. Relapse on the 21st day of disease. Temperature 103°F., vomiting. Fauces injected. Definite punctate erythema on the trunk. Was in an acute ward when the symptoms appeared.
2. J.A. Male. aet, 3 years. First attack mild. Relapse on the 19th day. Temperature 103°F at the beginning. Vomiting. General punctate rash. Adenitis. Double otorrhoea. Ulceration of faucial structures. Death nine days from onset of symptoms. Was in an acute ward.
3. G.R. Female. aet. 11 years. Relapse on 12th day. Temperature 103.6°F. Headache. Vomiting. Fauces injected. Punctate rash on the trunk. Was in acute ward.
4. E.W. Female. Aet, 6 years. Relapse on 23rd day. No vomiting. Fauces swollen, injected, with soft exudation on the tonsils. Recurrence of strawberry tongue. Was in convalescent ward.
5. A.F. Female. Aet, 4 years. Mild primary attack. Relapse on 24th day. Temperature never higher than 101°F. No vomiting. General well marked punctate rash. Later, developed adenitis and otorrhoea. Was in convalescent ward.

6./

6. W.D. Male. Aet 3 years. Moderately severe relapse on the 22nd day. Vomiting. Faint punctate rash on the trunk. Fauces injected. Well marked strawberry tongue. Afterwards developed double otorrhoea and erythema nodosum, was in acute ward.
7. M.H. Female. Aet. 10 years. Moderate primary attack. Relapse on 20th day. Desquamation very marked. No vomiting. Strawberry tongue. Adenitis. Punctate rash. Ulceration of the tonsils,was in acute ward.
8. M.S. Male. Aet 3 years. Mild primary attack. Relapse on 23rd day. No vomiting. General punctiform rash. Adenitis. Rhinitis. Was in acute ward.
9. E.B. Female. Aet. 10 years. Relapse on the 21st day. No vomiting. Temperature 100°F. Rhinitis. Fauces injected. Faint trace of albumen in the urine on the same day, twelve days later, haematuria, was in acute ward.
10. A.H. Female. Aet. 11 years. Fairly severe attack on admission. Relapse on 21st day. No vomiting. Punctate rash on the trunk, finely macular on the extremities. Fauces injected. Spleen enlarged. In acute ward.

11./

11. A.D. Female. Aet. 15 years. Mild primary attack. Relapse on the 18th day. No vomiting. Fauces injected, General punctate rash. Swelling and pain in the finger joints. Was in acute ward.
12. A.B. Male. Aet, 5 years. Relapse on 30th day. Temperature 102.4°F. Vomiting. Well marked punctate rash. Fauces injected, with some follicular deposit. Was in convalescent ward.
13. E.M. Female. Aet. 3 years. Relapse on the 27th day. No vomiting. No pyrexia. Well marked punctate rash on the trunk persisting for three days. Strawberry tongue, Was in acute ward.
14. B.E. Female. Aet. 6 years. Relapse on the 24th day. Vomiting. Temperature 102°F. Punctate erythema on the trunk. Fauces swollen and injected, with some follicular deposit. Tongue strawberry. Was in acute ward.

The secondary attacks were not as a rule severe, the temperature in most cases fell to normal inside a week. The one exception was the only fatal case, No.2. In this case, the typical primary attack, and the presence of definite desquamation left one no option but to diagnose relapse; the course of the case was that of angincus scarlet fever in its worst form. In Henoch's experience relapse does not differ/

differ in any essential point from the original attack, it may be more mild or more severe.

Von Jurgenson quotes Theodore Hose of St. Petersburg, who found that, out of seventeen cases, three only could be called severe. The rash in all instances was less marked than in the primary attack, and also, a feature that my cases illustrate, the primary attack is usually only moderately severe. Caiger, (35) on the other hand, thinks that the severity of the second attack is inversely proportionate to the first. He has rarely seen a fatal case, but they are frequently severe.

There appears to be unanimity of opinion with regard to the period at which relapses occur. All observers agree that the greatest liability is during the third and fourth week. In my cases the earliest day it was noted was the 12th, and the latest the 30th- the majority, however, occurred between the 18th and 24th day.

There is not much of interest about the actual Clinical features of relapse. The condition of the throat is indistinguishable from that of primary scarlet fever. The rash presents the same punctiform appearances, and its occurrence with desquamation is very characteristic. In my experience, the tongue does not shew the injection, with enlargement of papillae/

papillae that is so constant a feature of the initial attack, but in five of my cases it was denuded of epithelium, and the papillae were enlarged enough to justify the use of the term 'strawberry.' Vomiting, so frequent as an initial symptom of scarlet fever especially in children, was only present in six cases, in the other eight its absence was particularly noted. Theodor Hose also remarks that vomiting is not nearly so frequent a symptom as in a primary attack.

A great difference of opinion exists as to the true nature of this so-called relapse. Some maintain that it is a real relapse, in the same sense as we use the word in reference to typhoid fever; while others hold that it is a second attack of the disease. I think the question really comes to be, are these patients the subjects of an auto-infection, or do they become re-infected from the outside - from the other patients in an acute scarlet fever ward. If the latter be possible, it must be a point of some importance in the administration of infectious diseases Hospitals.

Henoch, in the New Sydenham Society's publication, gives his support to the theory of true relapse, pointing out the analogy between these cases, and the relapses so frequently met with in typhoid fever. He suggests that/

that the scarletinal virus had not been fully eliminated by the first attack, and that, consequently, relapse was inevitable. Dr. Hunter,[36] referring to a case occurring in private practice, comes to the same conclusion. Von Jurgenson thinks that a relapse can be explained by the supposition that the scarletinal toxin is able, by "flaring up afresh," to relight up either the whole disease, or a portion only of its symptoms, even in the convalescence.

Caiger, in Clifford Allbutt's system, gives it as his opinion that any distinction between the two sources of infection is purely arbitrary; a difference cannot be drawn between a true relapse and a reinfection. In both instances the patient has not acquired sufficient immunity to protect himself from another infection, either from his own person, or some outside source.

Dr. Hodgson [37] holds the same view. Both observers, however, evidently believe that relapses are much more common in hospital than in private practice, the infection constantly being augmented and renewed by the admission of fresh cases, so that any patients who have not acquired sufficient immunity are liable to relapse.

Which of the two theories is most frequently the correct one, it is very difficult to decide; arguments in/

in favour of both may be advanced. I have previously remarked that most of my cases occurred between the 18th and 25th day, and that most observers have had a similar experience. Does not this fact suggest that there is some inherent tendency for the disease to "flare up again" at this period? If it were a reinfection from without, why should the patient be most liable to it at one particular period? Caiger's explanation of a short lived immunity, would render them more liable to attack in the 5th or 6th week, just before discharge.

It is remarkable that the period for relapse and the period for nephritis, secondary throat, purpura etc., should coincide. I have seen the suggestion put forward in some paper that I have mislaid, that in relapse it was the specific infecting organism of scarlet fever, whatever that may be, that was responsible, while in the other group of complications it was septic organism that was present. This theory no doubt depends to a great extent on pure speculation, seeing that we do not know what the organism of scarlet fever is, yet it explains the phenomena so well, one is half inclined to think there may be some degree of truth in it.

Quite a number of cases are recorded in the Medical/

Medical Journals, in which well marked cases of relapse had occurred in general practice, and in which there could not have been any reinfection from another person. We cannot ignore the possibility of reinfection from furniture, bedding, etc., although I think such an occurrence is extremely unlikely.

If the immunity conferred by an attack of scarlet fever is so slight and so short-lived, that a number of cases become reinfected from an outside source and while still in hospital, why do we not more frequently see second attacks of the disease after the convalescence has been completed? I have not met a case yet in which there was reason to diagnose a second attack, apart from these so-called relapses. Henoch in all his experience only met one case in which he could reasonably conclude that, that had occurred.

On the other hand it is difficult to say that reinfection never takes place. Isolation Hospitals are considered by many to be responsible for its occurrence, as it is only since these institutions began to play the important part they now do in Public Health work, that relapses began to attract the attention of the profession. The explanation of this may be that they are less liable to be overlooked in Hospital, where the thermometer is used regularly through/

through the convalescence, and any noteworthy fact reported at once. Besides, when one man has a large number of cases under his care, he cannot help being struck by the comparative frequency with which relapse occurs, whereas an isolated case in general practice might only make one doubt the accuracy of the original diagnosis, especially as the primary attack is so often of a mild type. Still the fact remains that, for a long time, many competent observers had not their attention particularly drawn to the complication, and I should think it is unlikely that there has been any great change in the life history of the disease.

In the synopsis of cases of relapse that came under my observation, I have noted whether the patient was in an acute or a convalescent ward when the relapse occurred. This requires a word of explanation. It is the practice at Lodge Moor Hospital to pass each patient through two or more separate wards before they reach the final ward, occupied for a few days before discharge. The ordinary case of simple scarlet fever is allowed up in clothes on the 15th or 16th day, and is transferred to a convalescent ward any time from the 18th to the 28th day, the time spent in the acute ward depending on the nature of the case, and to some extent/

extent on the demand for empty beds. I know [38] that a somewhat similar routine is carried out in other hospitals, but whether with the same regularity I cannot say. In only three of the fourteen cases was the patient in a convalescent ward at the time of the relapse, although the average mild case is transferred about the time one would expect relapse to occur. I am afraid the numbers are too small to draw any conclusions from, but so far as they go, they seem to point to the possibility that patients are more liable to this complication in an acute, than in a convalescent ward.

Overcrowding and imperfect ventilation of scarlet fever wards is looked upon by some as a very potent cause of relapse. Dr. Millard [39] is of opinion that the segregation of a number of scarlet fever patients in one ward, leads to such an exaggerated virulency in the virus, that not only is the case mortality increased, but cases, infected by patients returning home from that ward, contract the disease in a more virulent form. As far as my own experience goes, patients contracting the disease in the Hospital do appear to suffer from it more severely, and shew a higher death rate. There certainly appears to be sufficient evidence to impress on one the importance/

importance of paying particular attention to ventilation, and, at the same time, of not allowing patients well advanced in convalescence to remain in acute wards. The French cubicle system, where each individual is quite isolated from his fellows, would of course be the ideal arrangement. We cannot be sure that some of the more frequent or more serious complications are not more likely to occur among patients living in such surroundings as I have indicated.

I personally have been unable to come to any definite conclusion as to reinfection or relapse, the evidence seems so conflicting. It is possible that, as Dr. Hodgson (37) suggests, both may occur.

Another subject, that has provided material for discussion among those interested in infections diseases, is Post-Scarletinal Diphtheria. This condition does not properly come under the head of either reinfection or secondary infection, yet the scarlet fever patient is usually said to be particularly susceptible, and therefore we may consider diphtheria as being on a different footing with regard to scarlet fever, than the other mixed infections, such as scarlet fever, measles, etc., which can only be accidents in the course of the disease.

The/

The Metropolitan Asylums Board Reports provide statistics from a very large number of cases, the percentage in 1902, 1903, 1904 and 1905 respectively, was 2.84, 2.32, 2.00, and 1.32. For a purely accidental occurrence, the fluctuation is not very marked.

Caiger (40) speaks of this condition as "the gravest complication to which the scarlet fever convalescent is liable." To shew the seriousness of the condition he quotes the M.A.B. Reports for 1893; there were 14.548 cases admitted to the different hospitals, the incidence of post-scarletinal diphtheria was 1.4%, and of these cases 58.3% proved fatal.

My own experience of this complication is very different from what Caiger describes. During a period of over two years here, I have not met with one case of post-scarletinal diphtheria, in which all the features we expect to see in ordinary diphtheria were present. I have seen cases with membrancus deposit on the tonsils, strongly suggesting diphtheria, and among them were some of these I have first described; in none, however, was Loeffler's bacillus found, and in none did any of the sequelae follow, that one would naturally expect to follow, if they/

they had been cases of true diphtheria. One may reasonably anticipate some paralytic sequelae, when, in the early stage, there is a continuous sheet of membrane covering the tonsils and uvula, and especially so, when there is evidence that the nasal mucous membrane is also affected.

I have also seen cases which, during convalescence, have exhibited features that impelled me to take a cultivation from the throat, and in a few instances typical Loeffler's bacilli were found. Yet I have experience of no case of this kind that later developed paralysis, such as one expects to find in a certain proportion of all cases of true diphtheria. I do not think that this absence of the frequent sequelae can be altogether explained by the fact, that antitoxin was immediately injected if the clinical aspects of the case in the slightest degree resembled true diphtheria.

The onset of signs of laryngeal obstruction, during scarlet fever, might well be taken as an indication that the condition was possibly complicated by diphtheria. By general consent, it is rare for the inflammatory changes induced by the scarletinal toxin to spread to the larynx; still, most observers grant that it occasionally does so. Henoch in particular pays considerable attention to the subject, he says that/

that it may occur in different degrees, froma simple hoarseness which is moderately common, to marked laryngeal obstruction with indrawing of interspaces and epigastrium, and inspiratory stridor. He advocates tracheotomy in these cases, and also in those in which - although the larynx is quite free - the swelling of the faucial and retro-pharyngeal tissue causes an actual obstruction. He has only seen one case recover after tracheotomy. Dr. Collie's experience is similar,(42) he says that laryngitis in scarlet fever is usually fatal and advises trachectomy when there is any "distinct difficulty in breathing." On the other hand, Haviland Hall(43) appears to doubt whether scarlet fever ever attacks the larynx, except when complicated by diphtheria.

I have performed tracheotomy four times for urgent laryngeal obstruction in scarlet fever, with one recovery, and in every case excluded diphtheria, as well as it is possible, by bacteriological examination. In none of the three fatal cases was there any appearance of membrane, either in the larynx or trachea, but marked thickening of the laryngeal mucosa, and the aryteno-epiglottidean folds. The ulceration of the tonsils, uvula, and in one case of the mucous membrane covering the tip epiglottis was extensive/

extensive. In all the fatal cases broncho-pneumonia probably was present before the operation, and appeared to be the immediate cause of death. One rather serious trouble arises from the ulceration that is apt to spread from the tracheotomy wound, the septic discharge seems to be responsible for it. It was much more difficult to control than the slight degree of the same condition, sometimes seen after tracheotomy for laryngeal diphtheria. I think better results might be obtained from tracheotomy in bad cases, if they were operated on before the onset of broncho-pneumonia. The postural treatment, of raising the bottom of the bed, which we use extensively here, is, I believe, of decided value in preventing the onset of this very fatal complication; by this means, if the child is at the same time turned half on its face, all the discharge tends to drain from the mouth and nose.

The organisms than may be found on cultivating the throat in scarlet fever, may be divided into three classes.

(1) Cocci, including streptococci and staphylococci; these are present alone, in the great majority of cases.

(2) Hoffmann's bacillus, first described in 1888.

(3)/

(3) Bacilli which, morphologically, are indistinguishable from Loeffler's bacillus, and which we must for the purposes of isolation accept as true diphtheria.

I have taken the results obtained from cultivating fifty severe cases on admission, and have found that in forty-four cocci only were present, in one instance Loeffler's bacillus, or at least an organism having the same staining reactions, etc., and in the remaining five bacilli were present conforming to the type described by Hoffmann; they are short, thick, straight bacilli, arranged often so as to form sets of two or more lying parallel, they stain very deeply with methylene blue, and shew no beading. A great difference of opinion appears to exist as to the true significance of these "pseudo-diphtheria" bacilli. Probably a number of different organisms have been described as such, and we must bear in mind that it is quite likely that some may be attenuated forms of the true Loeffler's bacillus; the fact that they are non-virulent does not appear to entirely negative this supposition. (44) (45)

Garratt and Washbourn (46) classify the bacilli that may be found in the throat of scarlet fever patient as follows:-

(1)/

(1) The Klebs Loeffler bacillus of true Diphtheria.

(2) Bacilli resembling above in cultural and morphological characters, but not pathogenic to guinea-pigs.

(3) Hoffmann's bacillus.

For practical clinical purposes the first two classes become one. From cultivations in 666 cases, admitted as scarlet fever and clinically suffering from that disease, they obtained the following results:-

A. Bacilli, morphologically resembling diphtheria, present in 8 cases or 1.2 per cent.

B. Hoffmann's bacillus present in 21 cases, or 3.2 per cent.

One of the eight cases, with diphtheria bacilli present, shewed signs of very slight ciliary paralysis, all the others made good recoveries. Only in one case did the clinical appearance suggest diphtheria, and this case alone received antitoxin, but no symptoms of diphtheria intoxication followed.

Dr. Syme (47) found that among 68 cultivations in early scarlet fever there were nine cases with short bacilli, and one in which the long bacillus was present; one only of the former cases shewed slight paresis/

paresis of the palate and loss of the knee jerks, this case had no exudation on the tonsils.

Sörensen's [48] experience is much the same. In 1547 cases admitted to Hospital, 38 or 2.5 per cent, had Klebs Loeffler bacillus present. He remarks on the small number of cases of clinical diphtheria breaking out under these circumstances.

In one particular, my results differ materially from those of Garratt and Washbourn. Hoffmann's bacillus was present in 10 per cent of the cases I cultivated. In most of my cases the attack of scarlet fever was a severe one. Is it possible, that an organism of this character is more likely to be present in the worst types of the disease, in which all the mucous membrane of the upper air passages is affected? In this connection the form of post-scarletinal rhinitis described by Todd [49] is very interesting; he isolated a bacillus resembling that of true diphtheria in all respects, including pathogenicity, but in no case was it accompanied by either faucial or laryngeal diphtheria.

I might restate my experience of diphtheria, associated with scarlet fever, in this Hospital. The true Loeffler's bacillus is occasionally found on admission, but so far, I have not known such a case to be/

be followed by paralysis. As regards the convalescent stage of the disease, I have seen several cases in which, on cultivating the throat because of some suspicious deposit, bacilli, very like the true bacillus, were present, but no sequelae followed. Needless to say, I have not known of a fatal result where all the clinical and bacteriological feature of diphtheria were present, in a convalescent scarlet fever patient.

How am I to reconcile these facts with the reports of the Metropolitan Asylum's Board, and particularly with the 58 per cent of deaths?

Several points have occurred to me as throwing some light on the subject. In the first place, there appears to be a general consensus of opinion that bad ventilation, over-crowding, and unsuitable surroundings favour the outbreak of diphtheria in scarlet fever wards. Caiger mentions the fact, that, in one Hospital he knows, cases are twice as common in some wooden huts as in modern brick-built wards, the huts however were deficient in floor space. At Lodge Moor we have modern stone wards, and also some wooden buildings, which are, however, of a particularly substantial pattern, with an efficient drainage system. Over-crowding is carefully avoided.

Probably/

Probably more important still is the situation of the Hospital. It is five miles from the centre of the city, over 800 feet above the sea level, and is exposed to an almost constant westerly wind, so that natural ventilation of the most perfect type is very easily attained.

Dr. Goodall (50) advances the opinion, that the special tendency of the convalescent scarlet fever patient to contract diphtheria has been very much exaggerated. He points out that the prevalence of post-scarletinal diphtheria closely follows the primary form, and its incidence is not materially higher among patients in hospital, than among the juvenile population outside. I am much disposed to look on Dr. Goodall's suggestion as providing the most plausible explanation, why this condition should, in my experience, be such a rare one. Five or six years ago diphtheria was epidemic in Sheffield, but lately it has not been at all common, while in its most severe forms it has been rare. During 1905 the notifications numbered 407; of these, 226 cases were sent to hospital, but only 143, or 63 per cent turned out to be true diphtheria. If there were the same number of mistaken diagnoses among the cases kept at home, it was not a heavy incidence for a city with a population of 450,000. In the year 1906, 189 cases were/

were treated. The fact also must be noted that in a number of the cases the diagnosis was based on the bacteriological examination alone.

What is the source of infection when post-scarletinal diphtheria does occur? I think that undoubtedly these occasional cases, that are admitted with bacilli present probably in a less virulent form, must be a great source of danger. It is now, I believe, a generally accepted fact, that Loeffler's bacillus may be found in the throats of persons exhibiting no clinical sign of the disease, and that these individuals are quite capable of communicating diphtheria to others, even in a very virulent form. The possibility of infection from attendants, toys, food, etc., must not be forgotten.

As to the measures that are most likely to prevent the outbreak of this complication, careful attention to the rules of hygiene, and a reasonable care in admitting fresh cases into the wards, seem to be the most important. The ideal procedure would be to make a bacteriological examination of every case on admission, this however is hardly called for, except when diphtheria is in any way prevalent in the district. The possibility of a rhinitis of diphtheritic origin, such as Todd has described, must not/

not be forgotten. Rigid isolation of all cases with suspicious bacilli is essential, notwithstanding the fact that, as Caiger remarks, "a known case of diphtheria in a ward rarely infects others." Although the so-called "short bacillus" appears to be usually innocuous, it is necessary to exercise great care, and when any doubt exists, treat the case as one of diphtheria.

When diphtheria bacilli are present in the scarlet fever throat, with exudation of a membranous character, I think one cannot avoid administering antitoxin.

BIBLIOGRAPHY.

1. Clifford Allbutt's System of Medicine, II, 158.
2. Nothnagel's Encyclopedia (Saunders), 495.
3. Bristol Medical Chirurgical Journal. March 1897.
4. Journ. Path. and Bactericl., March 1897.
5. Brit. Med. Journ. (1906) I., 421.
6. Lancet, August 13th, 1892.
7. Lancet, (1897) I., 1480
8. Lancet, January 20th 1894.
9. Henoch, Diseases of Children, New Sydenham Soc. 1887, page 221.
10. Sörensen, Ueber Scharlachnephritis, 316.
11. Nothnagel's System, 496.
12. Clifford Allbutt's System, II, 152.
13. Nothnagel's System, 520.
14. Transact. of the Path. Soc., (1877) 430.
15. Allbutt's System, II, 166.
16. Deutches Archiv. Fur Klin.Med. LXXVIII, 209.
17. Boston City Hospital Reports, (1899) 440.
18. Brit. Med. Journ., (1906) I, 1376.
19. Bulletin gen. de therap., (1904).
20. Allbutt's System, II, 154.
21. " " II, 157.
22. Wien Klin. Wchnschr., (1902) 41.
23. Berl. Klin. Wchnschr., (1902) 48, 49.

24./

24. Osler's Text book of Medicine. 79.

25. Reynold's System of Medicine, 165.

26. Lancet,(1902) II, 286.

27. Henock, New Syd Soc., 1887, 231.

28. Lancet, (1906) II, 1466.

29. Lancet, (1902) II, 286.

30. Lancet, (1903) I, 197.

31. Lancet, (1891) I, 658.

32. Lancet, (1893) II, 366.

33. Collie on Fevers, H.K.Lewis, (1887) 33.

34. Henoch, New Syd. Soc., 232.

35. Allbutt's System, II, 162.

36. Lancet, (1895) II, 445.

37. Lancet, (1903) II, 1258.

38. Dub. Journ., of Med. Sc., (1898) 324.

39. Public Health, Vol. XIII.

40. Allbutt's System, II., 159.

41. Henoch, New Syd. Soc., II. 217.

42. Collie on Fevers, H.K.Lewis.

43. Lancet, (1897) I, 363.

44. Muir and Ritchie, Manual of Bact.

45. Cobbet and Phillips, Journal of Path. and Bact., IV. 193.

46. Brit. Med. Journ., (1899) 206.

47. Lancet, (1895) II, 455.

48./

48. Ed. Med. Journ., (1899) 206.

49. Lancet, May 25th, 1898.

50. Lancet, (1896) I, 710.